BRAIN.

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Original Articles.

HEAT-CENTRES IN MAN.

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After my discovery of the seat of the cerebral inhibitory centres of sphincters, it occurred to me that by similar means I could find the location of the basal heat-centres. Transverse sections were made of the brain from before backwards, and when I divided the corpora striata, a temperature of 111½° F. was obtained.

In April 1884[1] I stated that heat-centres existed about the corpus striatum, which is true to-day. In July 1885[2] I also announced that the anterior part of the thalamus, when injured, caused an elevation of temperature due to augmented production of heat as shown by the calorimeter.

After the above dates, October 1885, Messrs. Aronsohn and Sachs[3] stated that injuries on the medial side of the nodus cursorius and beneath the corpus striatum caused an elevation of temperature with increased absorption of oxygen, increased exhalation of carbonic acid and augmented excretion of urea. It is true that they published a paper[4] (December 18, 1884, six months after my publication), on the elevation of temperature after punctures, locating the origin in the anterior part of the brain, a fact already discovered by Richet. These facts destroy the claims of Drs. Aronsohn, Sachs, and Richet to priority of discovery of the basal heat-centres.

[1] 'Journ. of Nervous Disease,' 1884. [2] 'Philadelphia Medical News,' 1885.
[3] 'Pflüger's Archiv,' 1885. [4] 'Deutsche Medicin. Wochenschr.' 1884.

Further, the determination by them of the increase of oxygen absorbed and of carbonic acid thrown off, with augmented excretion of urea, was no absolute proof that the elevation of temperature was due to increased production of heat: for D'Arsonval has shown that chemical processes do not give the exact measure of the production of heat, since the incubating egg in its first stage absorbs considerable heat and also oxygen, whilst it exhales carbonic acid. This absolute proof was only furnished by the calorimetric experiments.

There have been localised in the lower animals six centres, injury to which causes increased temperature. The cruciate about the Rolandic fissure, the sylvian at the junction of the supra- and post-sylvian fissures, the caudate nucleus, the tissues about the corpus striatum, a point between the corpus striatum and optic thalamus near the median line, and the anterior inner end of the optic thalamus itself. These centres have their own laws, which serve to distinguish one from the other. In a paper published by Dr. W. Hale White,[1] there has been collected a number of cases, which I have mainly drawn upon to support the theory of localised heat-centres in man. He rules out all cases where the cause is not purely nervous. Thus cases with bed-sores, where suppuration might cause fever, are excluded.

In support of the localisation of a heat-centre about the Rolandic fissure, the following case is quite definite:—H. W. shot himself with a pistol. Upon admission he was insensible, with a small triangular wound on the right side, between the external angular process and the top of the pinna, respiration irregular, 28–35, pulse 52, strong, regular, right pupil widely dilated, left strongly contracted, both insensible to light. Almost directly after admission the bullet was extracted. Immediately after the operation, the temperature was 99·2°, but it ran up in a few hours to 104·2°. It remained high, being half an hour before death 104·4°. Death ensued twelve hours after the operation. Upon post-mortem it was found that the bullet had not perforated the dura mater. The anterior extremity of the middle lobe of the right hemisphere was severely contused, as also the third frontal convolution,

[1] 'Guy's Hospital Reports,' 1884.

the damage affecting the whole thickness of grey matter. This case lends considerable support, taken in connection with the location of the cruciate centre in lower animals, that the parts about the fissure of Rolando, when injured, cause an elevation of temperature. In confirmation of this view, a remarkable case has been recently reported by Professor J. D. Blake.[1] F—— was found sitting by the roadside asking to be taken home. With slight assistance he was helped into a waggon, but upon trying to leave it three-quarters of an hour afterwards, he was unable to do so. He spoke, swallowed, requested water during the night, and asked for something to eat. In the morning of the next day he became stiff, his tongue was thick, he seemed stupid and drowsy. On the third day he was seen by Dr. Blake, who found him paralysed on the right side, and removed a spike driven through the skull into the brain. The spike was three and one-eighth inches long, and three-eighths of an inch in diameter. It was driven in till the head of the spike pressed firmly upon the scalp. Following its withdrawal was a small quantity of offensive decomposed blood and serum. After the removal of the spike there was right complete hemiplegia, sensation nearly abolished on same side. Cannot talk but understands, tongue runs to left side, answers questions by nods of the head, respiration slow, pulse full 94, temperature 103⅗°. On fourth day, pulse 98, temperature 104°, quiet, suffers no pain, swallows badly, slight muscular twitchings of the left side. On fifth day, profound stupor, takes nothing, pulse 98, temperature 104°, had convulsions, respirations 22, temperature in the evening 103⅗°, seems to be sinking. On sixth day, respirations 51, pulse very rapid, became convulsed, comatose, and died. Upon post-mortem it was found that the spike entered the skull through the left parietal bone near the median line, and one and a half inches from its junction with the frontal, passing obliquely downwards and forward, entered the left hemisphere just anterior to the Rolandic fissure, to open into it all the way down to the corpus callosum. The spike entered the lateral ventricle, the point slightly entering the intra-ventricular portion of the corpus striatum. In this case there

[1] 'Philadelphia Medical Times,' July 1888.

are four causes of high temperature: meningitis, putrid blood, striate injury, and lesions of the convolutions about the fissure of Rolando. It is not likely that putrid blood or meningitis would cause so great an elevation (104°), and the lesion of the corpus striatum was not extensive enough. There is every reason to believe that the Rolandic convolutions bore the greater part of the injury, and were the cause of the elevated temperature. The case previously related had a temperature of 104°. Bourneville reports a case of softening in the neighbourhood of the fissure of Rolando with a temperature of 105°.

Sylvian Centre.—In the cortex of cats there have been discovered the cuneate and Sylvian thermotaxic centres. Through the kindness of Dr. C. K. Mills, I have learned of a case which strongly supports the view of a thermotaxic centre in man about where I discovered the Sylvian. In the London 'Lancet,' Jan. 2, 1887, Dr. Page reported a case of depressed fracture of the skull, which was about the posterior part of the tempero-sphenoidal lobe, where a temp. of 105° F. existed. No other symptoms were present, and the patient was trephined on account of the high temperature. The operation caused the temperature to fall to nearly normal, not to rise again. The patient recovered.

Lesions of the Corpus Striatum.—Bourneville relates a case of a large patch of red softening in the left centrum ovale, and a recent patch in the right corpus striatum, where the temperature was 104·8° before death. Dr. White reports a case which undoubtedly shows the corpus striatum to be the seat of a thermic centre. W. C., æt. 6½, was admitted to the hospital. Before admission, he was paralysed on the right side, which came on suddenly, and was followed by incontinence of urine. On admission three months afterwards, he was found to be weak on the right side, never spoke, took no notice of things going on around him. For a month his temperature remained abnormal; but after that time had elapsed, the temperature rose to 102·4°, and the boy had a fit at the same time. About three weeks before his death he became completely unconscious, and remained so. Upon post-mortem no evidences

were found of tubercle, meningitis, tumour, or disease of the cerebral arteries. Just above the anterior commissure of the brain was a brownish patch about a quarter of an inch in diameter, so soft that it left a hole when the brain was cut. These patches were symmetrical both in colour, size, and position; well-marked grey discolouration was present in both crura cerebri at their anterior inner margin. This degeneration extended down the cord to the lumbar region. All the other organs in the body were perfectly healthy. The table gives the temperature of this remarkable case.

March 24 to April 3	Temp. range 95°—96°
April 3 „ „ 17	„ „ 97·4°—98·4°
„ 17 „ „ 24	„ „ 95°—97·4°
„ 25	Morning 101·2°, Evening 104°
„ 26	„ 97·4°, „ 99·2°
„ 27	All day 97°
„ 28	Morning 98°, Evening 100°
„ 29 to May 25	„ 96°—99°—100°
May 26	„ 100·2°, Evening 106·6°
„ 27	„ 105°
„ 28	Kept at 102° by sponging
„ 29	Morning 102·4°, Evening 99·2°
„ 30	„ 105° (convulsions)
„ 30 to June 5	102° by sponging
„ 6 „ „ 8	98°—100°
„ 8 „ „ 11	102°
„ 11 „ „ 16	103°
„ 17 „ „ 18	105° Just before death 107°, fifth exacerbation. There was wasting of muscles all over the body.

A similar wasting of the muscles is seen in animals whose thermic area in the corpus striatum is injured.

Dr. Bagojawlensky[1] has reported a case of elevated temperature where the only lesion was echinococcus cysts in the corpora striata.

Thalamic Lesions.—The pathological cases in this area are not very much to the point. Ollivier reports a case of hemorrhage into the optic thalamus and crus cerebri with a temperature of 102°. Dr. Beevor reports a case of a boy, æt. 11. Present illness came on five months ago. Four months ago he had a fit, afterwards paralysis of the muscles of the eyes, left arm, left leg, and left face. On admission,

[1] 'Centralblatt f. Med. Wiss.,' 1888.

all the above parts were paralysed. There was intense optic neuritis. He was deaf, and passed urine and fæces under him. Persistent priapism was present. The temperature the evening before death was 104°, just before death 107·4°. Upon post-mortem there was found an abnormal growth, three inches long and two and a half broad, starting from the upper surface of the cerebellum, extending forward between the optic thalami, flattening the corpora quadrigemina, pressing upon the right crus and the right superior cerebellar peduncle. This case lends some support, but not a very strong one, that the thalami are concerned in the elevation of temperature.

Subnormal Temperatures.—It has been observed in many of my experiments upon animals that after an irritation of basal heat-centres the preceding elevated temperature was succeeded by a temperature and a heat-production below normal. Dr. Farnham has noted in a girl æt. 3 years with typhoid fever a similar state of affairs. Through the period of low range of the thermometer, beyond a slight irregularity of the heart's rhythm, the child developed no unfavourable symptoms, but gained steadily in strength, slept well, and ate in a satisfactory manner. The table shows the subnormal temperatures, taken with a Hicks' thermometer.

Up to November 9th, the temperature is rectal, afterward axillary.

	Morning.	Evening.
Nov. 7	104·8°	105·8°
„ 8	103·2	106·2
„ 9	102·2	104·2
„ 10	101·0	104·4
„ 11	—	103·4
„ 12	101·2	103·0
„ 13	99·6	102·8
„ 14	98·8	101·2
„ 15	97·6	100·2
„ 16	96·6	98·8
„ 17	95·4	97·4
„ 18	95·4	95·2
„ 19	96·0	97·0
„ 20	95·4	96·2
„ 21	95·6	97·2
„ 22	95·2	97·4
„ 23	96·6	97·8
„ 24	96·6	97·8
„ 25	97·0	98·2
„ 26	96·4	98·4

In consumption the temperature, according to Dr. Williams, is as follows:

I. A wide range of temperature extending from 94° F. to 103·4° F.

II. An afternoon pyrexia.

III. Subnormal temperature in the early morning, indicating the collapse influence.

Convulsive Affections.—In another place I have advanced reasons for believing that the high temperatures of tetanus are due to an excitation of the spinal thermogenic centres, and not to muscular movements. In an analysis of 410 cases of chorea, Dr. Osler arrived at the conclusion that fever takes place when rheumatism sets in, when endocarditis becomes severe, and in some cases of chorea insaniens. There may be the most intense and violent movements without any rise of temperature, and a great majority of cases remain afebrile throughout. Certainly muscular movements have little to do with elevated temperature in chorea. In epilepsy Lemoine has made two hundred observations of isolated attacks, and arrived at the conclusion that the temperature rises with the attack, but falls rapidly afterwards, so that even in a quarter of an hour it may be a little below normal, but after two hours it was about normal again. The average rise is 1·2° F., and the rectal temperature very rarely goes above 102° F.

Tumours and lesions of the spinal cord, oblong medulla or pons varolii, are all usually followed by high temperature. These cases are explained as being due to a removal of the influence of the thermotaxic centres, allowing spinal thermogenesis to become exaggerated. Low temperatures here are due to excitation of the thermotaxic centres, their fibres, or to affections of the thermolytic apparatus.

High Temperatures.—There have been reported a series of cases where no adequate cause was found, but considering the extensive apparatus subservient to temperature, the lesion has probably been overlooked. Teale's case of injury to the spine with a temperature of 125° and Mahomed's of 128° are remarkable temperatures. These cases, if true, and I am inclined to

believe them possible, are somewhat parallelled by the experiments of Eulenburg and Landois upon the Rolandic centre, where they found a difference of 23° F. between the extremities of a dog. Hence it is quite possible that a similar high temperature might exist in man, after a disturbance of the Rolandic heat-centres. The rapid and high elevation of temperature often seen in malarial paroxysms indicate to my mind an action of the poison upon the thalamic centre, as it is the most sensitive heat-centre, gives the highest temperature, and the rise and fall correspond closely to the curve of a malarial paroxysm.

Diagnosis.—How far thermal observations will aid in the diagnosis of lesions of the brain is a difficult question to answer. If the other symptoms point to a lesion of the pons, certainly high temperatures will be an important aid. If the symptoms indicate a lesion about the corpus striatum, then high temperature with general muscular wasting will be corroborative.

These fragmentary observations show, in my opinion, that heat-centres undoubtedly exist in man. The task of establishing the localisation with all the required precision now devolves upon neuropathologists.

HALLUCINATIONS, AND THE SUBJECTIVE SENSATIONS OF THE SANE.[1]

BY D. HACK TUKE, M.D., F.R.C.P.

In the observations which follow I do not restrict my enquiry to the hallucinations of the insane. I include the subjective sensations of the sane.

Several cases, raising oft-debated but still undetermined questions in regard to hallucinations, have led me to think that they may be again profitably discussed.

Among those which arise are:—

1. Has any advance been made in regard to the nature and seat of hallucinations since the period when Brewster enunciated his famous statement, that *all* hallucinations involve the corresponding peripheral sense-organ?

2. More especially, does the discovery of sensory centres in the cerebral cortex disprove this position?

3. If lateral pressure on one eyeball so as to disturb the axes of the eyes causes double vision, is it a certain proof of the external presence of an apparent object? (Brewster's test.)

4. What are the tests of the retina being involved in visual hallucinations?

The case of Mrs. A., observed by Brewster, has become historical, and has reference to the third question. She had laboured under auditory hallucinations when he met her, and he thought it well to anticipate the occurrence of visual hallucinations, or as he terms them "spectral illusions," so as to put on her guard against taking them for realities. Now the test which he gave her was this. He told her "she might distinguish a genuine ghost existing externally, and seen as an external object, from one created by the mind, by merely pressing one eye (or straining them both) so as to see objects

[1] Read at the Psychology Section of the Meeting of the British Medical Association, Glasgow, August 10, 1888.

double; for in this case the external object or (supposed) apparition would invariably be doubled, while the impression on the retina created by the mind would remain single."[1]

On the fourth question, Huxley speaks almost as strongly as Brewster, when he is referring to the case of Mrs. A. and the spectral cat which she saw. Huxley says, "There can be no doubt that exactly those parts of her retina which would have been affected by the image of a cat, and those parts of her auditory organs which would have been set vibrating by her husband's voice, or the portions of the sensorium with which those organs of sense are connected, were thrown into a corresponding state of activity by some internal cause."[2] It is true that there is a saving clause in regard to the sensorium, but he evidently does not dissent from Brewster. As is well known, Mr. Huxley can speak from personal experience, which he has thus recorded. "I know not," he says, "if other persons are similarly troubled; but in reading books written by persons with whom I am acquainted, I am sometimes tormented by hearing the words pronounced in the exact way in which these persons would utter them, any trick or peculiarity of voice or gesture being also very accurately reproduced. And I suppose every one must have been startled, at times, by the extreme distinctness with which his thoughts have embodied themselves in apparent voices."[3]

It may be well for a moment to formulate the elementary principles of mental physiology in regard to sensory perceptions in health, taking vision as our illustration, and confining ourselves to the visual perception of external objects. For our present purpose, a very brief statement will suffice.

a. A distinct image is formed on the retina. Double vision occurs for a time when the convergence of the axes of the eyes in the object perceived is prevented.

β. In the sensorium a visual sensation is excited, the nature

[1] He adds that his test occurred to Mrs. A.'s mind when the hallucinations returned, but that "she was too well acquainted with their nature to require any such evidence of their mental origin; and the state of agitation which generally accompanies them seems to have prevented her from making the experiment as a matter of curiosity."—*Letters on Natural Magic*, 1832, p. 39.

[2] 'Elements of Physiology,' p. 240.

[3] Op. cit., p. 11.

of which is still very imperfectly understood. The ganglia of which the sensorium is composed are still undetermined. Whether the corpora quadrigemina are directly concerned with the visual sense appears to be uncertain.

γ. The sensory impulse is felt in the cerebral cortex, probably the occipital lobe, and according to Ferrier, in the visual centre in the angular gyrus, the molecular changes in which cause perfect visualization in the domain of consciousness—sensory impulses radiating to motor centres which are in relation to the muscles of the eye.

δ. Such changes in the cerebral cells—the results of the stimulus of an external object—persist as organic memory, stored up in the cortical centres.

If we adopt Dr. Hughlings-Jackson's mode of expression, we should have to say that in order to have a perception, or as he prefers to call it a vivid image of an object, say a brick, there is first the peripheral impression upon the retina, then secondly impulses pass through the lowest, through the middle,[1] and up to the highest sensory centres; from the most organised to the least organised; a multiplication of energy-liberations upwards, and consequently strong excitation and wide irradiation in the higher sensory centres. The physical condition in these centres, correlative with the red colour of the brick, has now been reached; but in order that the mind may become cognisant of its shape or the relation of its several positions one to another, which is acquired by ocular movements, there must thirdly be currents passing from the highest sensory centres to the highest motor centres, and from these downwards through middle and lowest motor centres to the peripheral muscles, causing movements of the eye-balls. The "vivid image" or mental state thus formed during the physical condition induced in the highest sensory and motor centres is, according to Dr. Jackson, "projected," because the lower centres are called into action, and thus it *seems* to form a part of the external world.

Passing on to the revival of sensory impressions in idea, we may say that—

1. Excitement of the cerebral cells which have undergone

[1] Sensorium?

these changes induces re-presentation of the original object in thought.

2. Such ideal revivification of the object induces the sensation, in however faint a degree, originally produced by the presentation of the external object.

3. What has been affirmed in regard to presentative and re-presentative sensations in visual perceptions, holds good of all the senses; and moreover the excitement of one centre tends to excite other sensory centres also, and, it must be added, sensori-motor action as well.

Now up to this point there is a fair consensus of opinion among psychologists, both in regard to (1) the physiology of sensory perception, and of (2) the ordinary healthy recalling in idea of sensations previously perceived, or of their combination in fresh forms; but a difference of opinion exists as to the extent to which the original sensory tracts are traversed —whether indeed there is any extension beyond the cortical centres.

As is well known, Herbert Spencer insists upon the identity in kind though not in degree of our primary sensations or presentative feelings and our ideas or re-presentative feeling: "The visual feelings are of all others the most easily reproduced in thought. The redness of a soldier's coat, the blue of the sky, the whiteness of the snow-covered ground, can be revived in consciousness at any moment with scarcely an effort, and with relatively great clearness. The glare of an electric light may be so vividly conceived as to produce something like a sense of dazzling."[1]

Or as Ferrier puts it, "The molecular thrill of present sensation extending from the peripheral organ of sense is in the ideal sensation revived; but," he adds (and this is the important point), "as a rule not so powerfully as to extend to the periphery."[2]

Professor Bain seems to believe (as Brewster did) that the whole sensory tract is traversed in simply recalling an object, distinctly, as, for example, the rose which we have seen.

In view of our existing knowledge of the functions of the

[1] 'Principles of Psychology,' vol. i. p. 229.

[2] 'The Functions of the Brain,' 2nd edit., p. 428.

cerebral cortex, there is no reason to adopt this opinion. The thought of an object already seen involves, as Dr. Jackson has maintained, the highest sensory and motor centres only, the reflex action being confined to these central links of the sensori-motor chain. "The central part only of the whole process which occurred in perception is done over again, and, the excitations being slight, the image arising is faint, and the lower centres not being engaged, it is feebly and indefinitely projected, and *seems* more part of ourselves."[1]

Next, as to the conditions attaching to *after-images* consequent upon impression received on the retina by luminous objects. We can try experiments at any time for ourselves in regard to these spectra, which we know to be retinal. For our present enquiry, the following facts are important.

Nothing is more certain than that (1) lateral pressure on the eye-ball or a multiplying lens fails to double them, although I have heard the contrary stated by some who have tried the experiment. Again, such spectra (2) always follow the movements of the eye. So long as the retina remains impressed with the image, the latter (3) obscures real objects when we open our eyes and look on a dark surface.

Here, then, we have reliable tests for ascertaining whether a visual hallucination involves the retina or not (Question 4).

The images of vivid dreams may cause a sense of "projection into space," and remain for some seconds after the dreamer awakes. A young lady, a relative of my own, has experienced this on three occasions. On one she saw her sister in a dream, and then awoke, when the figure remained distinctly visible for a short time at the foot of the bed; and on another occasion, a lady's maid rushed upon her in her dream: she awoke and saw the spectrum of the maid in the room.

Dr. Bastian informs me of similar experiences in his own case. It may be premised that it has several times occurred to him to awake, after going to bed, out of a short first sleep of about half an hour or less, with a feeling of bewilderment and forgetfulness as to locality, at the same time receiving an impression from a window opposite of its being very small and close to him: gradually, however, the visual impression

[1] 'Croonian Lectures," *Lancet*, 1884, p. 27.

enlarged and receded, till it was seen as when in a state of perfect consciousness; and as gradually too the feeling of doubt as to locality cleared up, and he became conscious of being in bed. It was on waking in such a condition as this (with face turned to wall) that he once imagined he was still sitting in his room reading, and that hearing a slight noise he turned round in his arm-chair, and looking towards the door saw a little fair-haired child standing with the handle of the door, just opened, in her hand. The child was the daughter of his landlady, and waking (perhaps from some noise) he seemed to see her so distinct and life-like for a space of several seconds, that a few seconds afterwards, on perfectly regaining consciousness, he was filled with astonishment to find it was all a vision, and that he was actually in bed. Notes of the occurrence were made in 1864, a few months after the circumstance happened. About twelve years afterwards he had a similarly distinct hallucination on waking out of a first short sleep. Then, as on the previous occasion, he had been for some time doing too much hard work. The figure he had seen in his sleep remained for some seconds after he awoke. In none of these instances were the tests just mentioned employed. The figure seemed however "projected," as in the case of the after-image of a luminous object.

I now proceed to mention several cases which bear upon the questions raised at the beginning of this paper.

1. A clergyman in Bethlem Hospital under Dr. Savage describes in a most graphic manner how he sees sentences addressed to him in legible type. He can distinguish the capital letters and see every word most distinctly. At first from his way of speaking, one supposes that he hears voices, but this is not the case. Thus he sees, not hears, himself addressed in the following terms: "I'll tell you where to go;" "I'm delighted with your misery;" "I'll stop up your passages." If he says what agony he is in, the words appear: "Yes, I'll give you worse;" "I'll make you break your head." "You must tear up all the Bibles and tracts." These sentences are continually communicated to him. One is, "Put up your spectacles, there's a good boy." The patient on telling me this last command, not unnaturally asks, "Can the brain do this?"

I have recently received the following short letter from him: "I do not think that I can add anything to what I have already said about my case, and can only therefore repeat that I am suffering from the never-ceasing presence in my head of *visible words*, and that, every moment. They are of a threatening character, such as 'I will give it you;' 'I have not done with you yet;' 'I will strike you with sores;' 'I know how to tease you;' and various other sentences too numerous to mention. Pray tell me whether you have heard of anything like this before? My agony is unceasing, and I cannot sleep." When his hat was one day blown off by the wind, he heard or rather saw the exultant words "I did it!"

This is the second attack, and has lasted five years. Twenty years ago, after having suffered in the same way for some years, he was operated upon by Sir H. Thompson for stone, and the result was instant recovery. The earliest symptom had been renal colic, followed by depression, and after a short time the appearance of words written in his brain. It should be added that Dr. Savage has requested Sir Henry Thompson to examine him during this second attack, but without any result beyond finding enlarged villi on the surface of the bladder. The instant disappearance of the visual hallucination after the operation would seem to show that, apart from the mental depression which might be referred to his gouty condition, the peripheral irritation of a calculus in the bladder acted on the cerebral centres and induced visual hallucinations. Did this act by inhibiting the highest mental level and allowing the lower level only to act? I conclude that in this patient's case the retina is not reached, for the words do not give the impression of being "projected," but seem to him depicted in his brain, and, as regards the effect of pressure on one eye-ball, no doubling of the words is caused, and he says they do not follow the eye.

In his case, then, the subjective image (1) is not projected, (2) is not doubled by pressure, (3) does not follow the movements of the eye, (4) does not cover objects, (5) is the same whether the eyes are open or shut.

2. The next case (H. J.) is that of a young man, also in

Bethlem Hospital, and is of special interest, because the visual hallucination of which he is the subject is a fixed one, a somewhat unusual circumstance. He has for a long time seen the figure of a man which he describes with great clearness, who is dressed in a blue coat, stands upon some scaffolding, having his left arm behind his back, while the right beckons the patient to him. Although he never speaks, the patient feels impelled to go in the direction the figure indicates by his hand, and this actually led the patient on one occasion to wander about till he was taken up by the police as a wandering lunatic, and it was on the same day I examined and certified him.

As this youth was quite willing to answer questions or try any experiment, a better case could not have presented itself, for trying the effect of altering the axes of the two eyes by pressing on one of them while the visual hallucination was distinctly in sight. Having first tested him with a picture hanging on the wall opposite, I found that under these conditions he at once said he saw two pictures instead of one. I got him to look steadily at the man in blue on the scaffolding, and, when he saw him before him as clearly as the picture on the wall, I pressed one eye in the same way as I had done before. Had he said he saw double, his reply would not have been conclusive; but as he said the figure remained single, I have no doubt that this was so, and that his case may be taken to show, so far as one case can do so, that subjective visual sensations are not doubled by interfering with the axis of vision. I repeated the experiment subsequently, with the same result. The hallucination became gradually fainter and fainter, and he ultimately recovered. It was interesting to watch the fading away of the image. He had ceased to believe in its reality long before it vanished. Here the image (1) was projected, (2) was not doubled by pressure. I have also no doubt, but this was more difficult to prove, it (3) followed the movements of the eye, and (4) was obscured by external objects.

3. The third case is that of a patient attending one of the London hospitals, about whose mental condition I was consulted some months ago. He complained of constantly seeing before

him the head of a dog. On examining him, I found that he was labouring under suspicious symptoms of general paralysis, but very intelligent in conversation, though losing his memory. Desirous to discover whether I could double the spectral object, I carefully tested him. He declared that when I pressed on one eye-ball he saw two dogs, but I was not convinced that he distinguished between the dog's head and the objects in the room, especially as he saw it distinctly only when he looked at the fire. On a subsequent occasion my doubts were confirmed. I cannot therefore allow this case to count as an exception to my experience in other cases. It is probably a case of illusion. An illusion may be doubled.

4. I pass on to a remarkable example of visual spectra in a perfectly sane person, upon which the above-mentioned experiment has been repeatedly tried, and with the result that no doubling the spectra took place.

Thanks to the courtesy of Dr. Lombard, of Norwood, the author of the well-known work on the 'Regional Temperature of the Head,' I am enabled to mention his own personal experience. Those who know his writings well know the conscientious accuracy which characterises them. The case is so conclusive and is so interesting on other grounds, that I must mention some of the particulars, especially the discovery of his singular power.

He had for some years occasionally seen images of persons and things which he had been attentively regarding, but he took little notice of the circumstance, and they speedily disappeared from the field of vision. On the 3rd of December, 1862, however, a much more decided visualization occurred. He was then in the army, and stationed on the west shore of Maryland. A military man, his daughter, a naval officer, and Dr. Lombard were seated in the verandah of a house which stood within a stone's throw of Chesapeake Bay. The girl sat on the second of the steps leading down from the verandah. He himself was on the same step at the other end, there being about five feet between them, and her profile being clearly in view. For no particular reason he began to stare at her, at the same time concentrating all his attention upon the features. At the expiration of about 20 minutes, he turned his eyes

towards the bay simply to relieve them, when he saw before him the image of the girl very distinctly, and also in like manner all the objects that had been within the range of his vision. The image began almost at once to fade, but he found that by fixing his whole attention upon it that he could retain it. To this power I particularly draw attention. He next looked over her head towards a wood of Southern pine-trees, and again saw the image. He had repeated this experiment four or five times when the girl turned her head towards him, and asked why he was staring at her so. Dr. Lombard described what he had seen, when to his surprise she laughed and told him he was only a beginner, and that he ought to be able to obtain in 20 seconds what had taken as many minutes. She then led him on the verandah to a point where the moon shone brightly, and asked the naval officer to take note of the time occupied in the experiments. She told Dr. Lombard to keep everything but herself out of mind, to look her full in the face, and then to run his eyes up and down her figure. In a few seconds she called "Time!" and told him to look towards Chesapeake Bay, directing him to regard the image as attentively as he had regarded her previously. He then saw the image very distinctly. The time was 22 seconds. In the course of the same evening many more experiments were made. In reply to her father and Dr. Lombard, she said that she had discovered that she possessed this power through a schoolfellow two years before, who possessed the same faculty of voluntarily recalling spectral images. Dr. Lombard asked her whether this power would last any considerable length of time. She said she could only answer for two years from her own experience, upon which the naval officer spoke up, and said that he could answer for nearly fifty years, having exercised it himself since he was fourteen or fifteen years of age.

Here then were four instances,—Dr. Lombard, the naval officer, the young lady, and her schoolfellow—who could by intense mental attention recall figures which they had once succeeded in fixing on the mental tablet. I may say, that Dr. Lombard never sees faces other than those which he has known and regarded attentively.

Is there conclusive evidence that Dr. Lombard's "faces" in-

volve the retina? One important test which was tried in his case —namely, whether the spectral figures moved with the movements of the eyes—yields an affirmative reply. If Dr. Lombard immediately after receiving the impression of a face brings up the image on the very spot, it will appear at nearly the same distance as the reality, and it will follow the motion of his eyes. This happened a short ago at Prof. B——'s house at Hanwell, where Dr. Lombard took (in photographic language) Dr., Mrs., and Miss X—— together, and each separately. At his own house in Norwood, at the present time Dr. Lombard sees them as if at a great distance, although perfectly distinct.

I enquired of Dr. Lombard whether the spectra concealed real objects in the room, or whether, on the contrary, he saw objects clearly through them. He says that he never sees a real object through a *good* image, but at times he has thought he could see more or less through an indistinct one. The concealment of external objects by ocular spectra is natural, and Brewster in commenting upon Mrs. A.'s case explains it by the recognised laws of optics. He held that this fact proves that the revival of an image does actually involve the retina. It is on record, however, that a gentleman who after waking from a dream saw the imaginary figure of which he had dreamt, particularly noticed that it made no difference in his seeing real objects behind the spectre. Can it be that when an object which has once been depicted on the retina is resuscitated as in Dr. Lombard's case, the image appears on the retina; but that when a purely fanciful picture, born of the fancy or of dreams, appears in spectral form, the cerebral visual centre, not the retina, may be the seat of the revived impression? Mr. Sully suggests to me that this might depend, not on the periphery being involved, but on the form and persistence of the image, which would make simultaneous attention to real objects impossible. Brewster's Mrs. A. saw the spectra of her husband and her cat, and they obscured her sight of objects in the room, but I am not able to say whether this occurred when the purely imaginary phantoms she describes appeared, nor can I say whether the visual hallucinations of the insane generally obscure real objects.

The power of transferring a single person or a group to another place, described by Dr. Lombard, is very remarkable He states that it requires a very strong concentration of the attention indeed.

Again, if two objects are very much alike, he can see their mental images together; but if he looks at one and tries to put the image of the other beside it, he will see only the reality. The other is invisible, if the resemblance be perfect, or it is a hazy object if there be some difference between the two.

I should say that the colours of the original objects are perfectly preserved in the spectra.

It must also be observed that Dr. Lombard never experiences any pain in his eyes after recalling spectral forms.

Before passing to another case of visual spectra, I would note that the revived images of Dr. Lombard (1) are projected, (2) are not doubled by pressure on the eye-ball, (3) follow the movements of the eye, and (4) cover external objects.

5. I have a young man under my care at the present time who labours under the delusion that he is dead. When I last saw him, he said he had distinctly seen the devil on the way. I asked him to draw the figure, which he immediately did, as you see here.[1] Roughly drawn as it is, it has evidently been done with conscientious accuracy. As he no longer saw the spectre I could not try any experiment upon the eyes. In his case the hallucination must have originated in the cortex, and not in the visual organ; just in the same way as an auditory hallucination must have had a cortical and not a sense-origin when a City man having lost his property becomes insane, and hears after a time a voice saying to him, "You are ruined." This is the sequence of events in insane hallucinations generally, but we cannot always retrace their history as in this instance.

6. A London physician called upon me just after this patient had drawn this figure,[1] and it elicited the interesting observation that both he and one of his boys occasionally perceived ocular spectra. They afforded good examples of what Galton

[1] The drawings were exhibited at the Lecture.

has called "visualization." The boy sees in a lane at a particular spot the figure of a little old woman, and is so accustomed to it that he mentions it in a matter-of-fact way without believing it to be real. His father, when a child, used to see a spectral figure in his bedroom, and can now resuscitate it with great clearness, as he did for my benefit when he described it to me. The room came up before him, and the figure in one corner, which was not in the direct line of vision but on one side. The moment he turned his gaze to this corner it vanished. He could only see the image when he looked in the same direction as that in which he looked from his bed when a child. While he did so I got him to press on the eye-ball of one eye, but no doubling of the image happened. On the other point whether the spectrum followed the movements of the eye, he says it does so. In his case, then, the image is (1) projected, (2) is not doubled by ocular pressure, (3) follows the motion of the eyes, and (4) covers external objects.

7. Of visual hallucinations in another person of perfectly sound mind, I had a very good example in a late medical friend, who was not in the least troubled with his spectral visitants. At my request he sketched some of them, and being a skilful draughtsman he made some striking portraits, which show that as in Blake's case he saw them as distinctly as if they had been actually present. [Drawings shown.] Unfortunately no experiments were made as to pressing the eye-ball, or observing whether the figures moved with the movements of the eye, &c.[1]

As I have, in the foregoing cases, given my own experience as to the non-doubling of subjective visual images by disturbing the axis of vision, it is only right to state that Despine of Marseilles records the case of an insane patient who had a visual hallucination of the Virgin Mary. He tried the effect of pressure on the globe of one eye, with the result, he says, that she saw two Virgins.

[1] I have thought that a few illustrative cases would be more instructive than the large number it would be easy to bring forward, but which would obscure the salient points I wish to keep clearly in view. I hope, however, at a future time to utilize the examples of hallucination of which I have notes, and which I shall continue to collect.

Again, Professor Ball of Paris obtained the same result by a like manipulation in a female lunatic who saw visions.[1]

Lastly, experiments by Binet and Féré in Paris on hypnotised subjects may be cited in support of the same experience. Time will not allow of my quoting these interesting experiments, but it is impossible to ignore them in any study of the nature and seat of hallucinations. Great credit is due to these observers for the extremely ingenious experiments referred to, and for the care they have evidently taken to avoid sources of fallacy. Whether, in so difficult an enquiry, they have always succeeded may, perhaps, be questioned; but I shall assume that the facts are exactly as they are described. It is only necessary to recall one instance. During hypnosis, a portrait is, by the suggestion of the experimenter, made to appear on a piece of cardboard. A visual hallucination is thus excited, and, moreover, it is located in a particular spot. Now it is this element in the experiment—the localisation of the hallucination on an external surface—which renders the results arrived at inconclusive in their bearing upon the point now under consideration, although important from other points of view. The observers found that every test which proves an object to be actually perceived applied to the hallucination which they had induced. A screen, or closing the eyes, rendered the hallucination invisible, and it was doubled by lateral pressure on one eye-ball, or by a prism. It could be seen reflected in a mirror, and the use of a spy-glass was accompanied by precisely the same effect as when applied to any object in the room. The glass had to be focussed for the imaginary as well as the real object.

Here, then, hallucination and actual perception appear to be proved to be identical. The result seems altogether opposed to what has been maintained in this article.

At first I was at a loss to reconcile this result with my own experience outside the phenomena of hypnotism. But further consideration satisfied me that the exception is more

[1] 'Leçons sur les Maladies Mentales,' p. 74. The case is complicated, however, by the patient being at the time in a state of artificial somnambulism. The explanation is probably the same as that suggested in those reported by Binet and Féré.

apparent than real. It is not denied, it is expressly stated, that the essential conditions of the success of this experiment were: an external object actually in sight, and one occupying the same position as the induced hallucination. One circumstance alone suffices to indicate the presence of conditions which remove the phenomenon from the range of ordinary hallucinations. When the card upon which the imaginary figure was depicted was turned round the "hallucination" disappeared; if inverted, the fictitious portrait was inverted also. Hence it is evident that the *point de repère*, as Binet terms it, and the suggested hallucination have become so completely fused and associated in the subject's mind by suggestion that they are inseparable. What the hypnotised subject really sees doubled by lateral pressure, what is suppressed by closing the eyes, and so on, *is* an external object, and therefore subject to the conditions attaching to all real objects within the range of vision. On reflection, therefore, I do not find in the interesting and important experiments of MM. Féré and Binet any exception to the results arrived at on other grounds in this paper. And, if I understand M. Binet correctly, I think his position scarcely differs from mine. The explanation of the apparent discrepancy is to be found in the surmise I made in the case of the patient who saw a dog's head when he looked into the fire. It is an *illusion*, not a hallucination—a change wrought in the appearance of an object by the fancy. It need hardly be said that an illusion would obey the same laws as an ordinary object of perception. In connection with this aspect of the question, it is necessary to find out, what seems to have escaped observation, that most of those who have referred to the Brewster test of objectivity have made a singular mistake in regard to it. In fact, they have completely reversed his formula. As already fully stated, his test was intended to distinguish between objective and subjective visual sensations. In the former case, the image would be doubled by lateral ocular pressure; in the latter, not. Remove the external stimulus, and doubling the image is no longer possible. So said Brewster; but the writers I refer to represent him as holding that mechanical pressure of the eye doubles the retinal image induced by

central excitation—a hallucination. Thus M. Binet writes: "Brewster performed this experiment on a subject who had visual hallucinations, and pressure on the eye caused a duplication of the imaginary object."[1] Brewster's intention, on the contrary, was to prove by non-duplication that the figure seen was a hallucination, and not a ghost. Again, I am surprised to find M. Déspine writing: "Brewster noticed that, in destroying the parallelism of the two eyes during a hallucination of sight, by pressure exercised on the external side of one of them, the patient upon whom he experimented saw the object of his hallucination double, exactly as we see a real object when we squint."[2] Déspine then mentions the already-referred-to case of his own in point, and it must count as one apparently opposed to my own experience. But what I am concerned to show now is that Brewster would have declared that the Virgin which M. Déspine's patient saw was real instead of being a fancy, from the very fact that the figure was doubled. It is still more surprising that so acute a mind as Mr. Gurney's should have misunderstood a British writer's argument on this subject. "The opposite party" (to Esquirol) "pointed out," he says, "that visual hallucinations often vanished when the eyes are closed; or, as Brewster first observed, that they may be doubled by pressing one eyeball."[3] Unfortunately, the false impression Mr. Gurney received from the study of Brewster, invalidates, to a certain extent, his treatment of the nature of visual hallucinations. In short, he, like many others, entirely confounded the tests of whether or not an apparent object is actually external to the observer, and whether the retina, as well as the visual centre, is involved in a given case of hallucination of sight.

It is difficult to conceive how the fact can be otherwise than Brewster stated, seeing that the reason why one external object is doubled by the proceeding in question is so obviously due to preventing the simultaneous action of corresponding points of the retinæ of the two eyes. The evidence ought,

[1] 'Annales Médico-Psychologiques,' Nov. 1881.

[2] 'Animal Magnetism,' International series, 1887, p. 227. As a matter of fact, Brewster only advised Mrs. A. to try this experiment, but she omitted to do so.

[3] 'Mind,' April, 1885, p. 168.

therefore, to be overwhelmingly strong in proof of the doubling of subjective images.

I demur to the axiom which has been laid down, that an imaginary object is perceived under the same conditions as a real one. I believe they differ in at least three particulars. First, in the effect produced by ocular pressure; secondly, in the behaviour of the image during movements of the eye; and thirdly, in the result of interposing an opaque screen between the observer and the object alleged to be seen.

I should substitute for this axiom the following: An imaginary object can be perceived under the same conditions as after-images. When this happens, the retina is involved. It would be extremely interesting to know whether, in cases of optic nerve atrophy associated with hallucinations, any tests have been employed to ascertain whether the conditions present were the same as those observed when the retina is known to be the seat of after-images.

I proceed to state Brewster's position in regard to the revival of sensory impressions in idea, and also visual hallucinations (Question 1).

"I propose," he says, "to show that the 'mind's eye' is actually the body's eye, and that the retina is the common tablet on which both classes of impressions are painted, and by means of which they receive their visual existence according to the same optical laws. Nor is this true merely in the case of spectral illusions; it holds good of all ideas recalled by the memory or created by the imagination, and may be regarded as a fundamental law in the science of pneumatology. . . . Spectres conjured up by the memory or the fancy have always a 'local habitation,' and appear in front of the eye and partake of its movements exactly like the impressions of luminous objects after the objects themselves are withdrawn." Thus Brewster asserts that not only spectral illusions (hallucinations), but ordinary mental pictures or ideas, are impressions on the retina, differing only in the degree of vividness with which they are seen, "being rendered more brilliant by some accidental and temporary derangement of the vital functions."[1]

[1] 'Letters on Natural Magic,' p. 49.

Dismissing Brewster's opinion so far as it relates to ordinary mental picturing of objects in idea, and confining myself to his dictum in regard to visual hallucinations, or, as he calls them, "ocular illusions," I would place in direct antithesis to it that of Esquirol, with whom Falret, Lélut, and other high French authorities concurred. Now Esquirol asserted that "Hallucination is a cerebral or a mental phenomenon, produced independently of the senses." . . . "One can only conceive the existence of this symptom by supposing the brain to be excited by a certain cause. . . . The images, ideas, notions, which appear to form part of the functional change in the senses present themselves to the mind with the same characters, and have the same seat, namely, the brain."[1] In short, according to Esquirol, the sensory nerves and peripheral sense-organs have not their functions called into requisition at all.

Well, if this is too unqualified a statement, as no doubt it is, for surely some hallucinations are due to peripheral pathological changes, it seems to me to be nearer the truth than the opposite extreme advocated by Brewster. For how can it be universally true when hallucinations of a special sense occur in the absence of the organ? Thus, visual spectra or hallucinations, may arise when the optic nerves are atrophied. Esquirol records the case of a Jewess who, although totally blind, laboured under extraordinary hallucinations of sight. She died, and he found that both optic nerves were atrophied from the point of their decussation to that of entering the eyes. In this instance, as he observes, the transmission of visual impressions to the retina was impossible.

Another patient under Esquirol had visual hallucinations, and became blind for seven years. Did this circumstance made any difference? Not the slightest. The hallucinations continued in full force all that time. He died, and Esquirol found the optic nerves of a grey colour, in fact, like wet parchment; flattened and atrophied throughout their whole course.[2]

These are merely illustrations of the instances in which sense-impressions have been capable of revival, although the

[1] 'Maladies Mentales,' vol. i. p. 200. [2] Op. cit. vol. i. pp. 187–8.

peripheral sense-organs have not been able to perform their functions. We are therefore driven to conclude that it is not absolutely necessary in even vivid hallucinations that the latter should be involved in their production. Stored up sensory images in the cortical centres may suffice (with or without the participation of the sensorium) for this revival. The increased knowledge we possess of the former supports this view.

There must, however, be sensory images to recall. On enquiry of Mr. Buckle, B.A., the Superintendent of the Wilberforce School for the Blind at York, who kindly investigated the subject, he informed me that those pupils who have no remembrance of having ever seen, never had had dreams of seeing objects, or experienced visual hallucinations. On the contrary, they were inclined to doubt the veracity of any blind persons who asserted that they had experienced them. Mr. Buckle found that some of the pupils who dreamed of domestic animals, dreamed of them as talking like Æsop's characters. I may add, that hallucinations of touch are by no means uncommon, pupils stating that their hands feel as if they were holding a ball or apple.

Here must be noted the familiar illustration of the patient who, although his leg is amputated, has vivid sensations of pain in his toes. No one doubts that the brain (sensorium or cortical sensory centre) may be the seat of a sensation which is mistakenly referred to a peripheral nerve-ending or sensory organ which no longer exists.

Whether, as a general rule, the peripheral terminations of the sensory nerves are reached in hallucinations is, however, another question.

The ordinary view is that promulgated by Carpenter, namely, that there is a backward current through "the nerves of the internal senses" to the sensorium, exciting sensations there precisely identical with those experienced when external objects are perceived, and the sensorium is reached through the nerves of the external senses.

According to Dr. Hughlings-Jackson, when there is distinct subjective perception of an object as in vivid hallucination, there is a stronger discharge of the same nervous arrangements

of the highest centres as in actual perception of an external object, so that the middle and then the lowest centres are overcome.[1] From his standpoint, it does not follow that because a patient labours under a hallucination, that that particular part of the cortical centre concerned in the special sense involved is diseased. Visual perception, and retinal impressions are represented in the highest cerebral centres, and when we recall to memory an object, there is not only the "purely psychical state," but a correlative physical state, namely a discharge of some nervous arrangement of our highest centres representing parts of the body, including impressions on the retina. Dr. Jackson's well-known theory of nervous discharges in epileptic fits accounts for the visual or auditory hallucinations which occur at the commencement of some of these attacks, as being mental states which "arise during slightly raised discharges of healthy arrangements untouched by the epileptic discharge.[2]

Discharging lesions of the cerebral cortex in epileptic attacks when accompanied by visual auditory or other aura, constituting one form of hallucination, bear so far upon this theory that they point to the origin of some hallucinations in the cerebral cortex and therefore disprove Ritti's position, based on the cerebral physiology of Luys, that all hallucinations originate in the basal sensory ganglia. It does not, however, follow that the impulse does not reach the peripheral sense-organs. I will briefly refer to an unpublished case of a female patient at the National Hospital for the paralysed and epileptic, under Dr. Gowers, as an illustration of the foregoing, kindly placed at my disposal, by Dr. Wilson of Wolverhampton who made the autopsy.

Six weeks before her admission, and three weeks after her confinement, she was standing and looking out of the window, when she suddenly heard a dreadful noise "like the clicking of machinery." She fell, losing consciousness and becoming convulsed. A fortnight afterwards, she had five similar attacks. The patient had a history of syphilitic sores, rash, and sore throat. For this she was treated by Dr. Gowers, and improved. After leaving the hospital, however, the fits returned,

[1] "The Croonian Lectures," 'Lancet,' 1884, p. 24. [2] Loc. cit.

accompanied by the same auditory hallucinations as before. There were no more seizures, but she died not long after, conscious to the last.

The autopsy revealed a tumour in the right superior temporo-sphenoidal convolution, about one and a half inches in diameter. For my present purpose, there is no need to describe the tumour or its surroundings in detail further than to add that it involved the middle temporo-sphenoidal convolution. The basal ganglia were quite healthy.

My attention was directed to this case by Professor Horsley, who, I may mention, not only holds that the cortical sensory centres would amply suffice for all sensorial hallucinations, however vivid, but hesitates to admit that there is any certain proof that in hallucinations, a current or impulse ever passes backwards down a sensory channel to the sentient nerve ending.

A case of epilepsy under Dr. Hughes-Bennett's care may also be mentioned. The patient had received a severe blow over the region of the angular gyrus some years before. A bright red flash of light usually preceded the fit. This was followed by visual hallucinations of different objects, judging from the manner in which he acted and spoke. These symptoms disappeared after an operation performed by Mr. Pearce Gould at the spot indicated by an old cicatrix. In his usual state the patient enjoyed normal vision.[1]

The reference to Dr. Hughlings-Jackson's views already made will indicate the mode in which he would explain the occurrence of hallucination in these cases.

It may be urged in favour of the Brewster doctrine that those patients who suffer from auditory hallucinations stop their ears with tobacco, etc.

It so happens that my attention was first definitely directed to the subject of hallucinations at the York Retreat, where my room looked into an airing court in which a male patient stood, day after day, carefully stopping his ears with his fingers. I was aware that he had auditory hallucinations, but it seemed to me extraordinary that as these were of subjective origin he should gain any relief from closing the outward ear

[1] 'British Medical Journal,' Jan. 1, 1887.

From that time I have endeavoured to ascertain from patients who hear voices whether any such relief is afforded by this proceeding, or again whether those who labour under visual hallucinations derive advantage from shutting their eyes.

It is indeed possible that no more relief is derived from the actions of patients in this respect, than can be accounted for by their naturally imagining that such relief can be obtained by shutting out what they regard as external influences and agents. There are cases, however, in which a local affection of the peripheral termination of the sensory nerve is the point of departure of the hallucination, and in such instances pressure on the ear, for example, by the fingers may afford some temporary comfort.

Again, in the case of auditory hallucinations of unquestionable cerebral origin, there was pain felt "in the ears." The patient was fresh from a three days' revival in London, under the auspices of the Established Church, and was brought to me hearing voices sending her to hell, along with distinct pain, as I have said, in the ears.

In Sir David Brewster's case (Mrs. A.), mention is likewise made of an uncomfortable sensation felt in the eyes when she saw phantoms. Again, it is so mentioned in Dr. Lombard's case.

Lastly, in some experiments in Hypnotism in Paris, it is stated that when visual hallucinations were induced, the subject complained of pain in the eyes. I do not wish to put any of these instances out of court; I only wish to point out how easily a person may refer to the periphery, through association of ideas with certain parts, that which does not extend further than the sensory centres.

Indeed, the fact that the general sensitiveness of the eye may be increased in the hypnotic subject of a visual hallucination, just as happens when a real object is waved before the subject's eyes, has been pointed to by M. Féré as a proof "that visual hallucination excites the visual centres."[1] He suggests that there are sensory centres common to the organs of the senses and to the integuments which cover them.

On the whole, then, I should say this of Brewster's position:

[1] 'Animal Magnetism,' p. 259.

that although it no doubt holds good in many instances, it is certainly not essential to the explanation of all hallucinations. As respects Esquirol's position, while it goes quite too far in ignoring the occasional origin of hallucinations in the sense-organs themselves, it seizes the important truth of the affection of the brain itself in hallucinations. Still, this great alienist could not bring to the support of his doctrine the knowledge we now possess of the functions of the cerebral cortex, and in answering the question, Does the discovery of sensory centres in the cortex disprove the position that all hallucinations of the senses involve the peripheral sense-centres? I should reply that it decidedly helps us to deny that such is absolutely necessary.

There is another theory of the pathogeny of hallucinations now in favour in France, based by Ritti upon the cerebral physiology of Luys which differs from that of Esquirol as also Brewster, and teaches that all peripheral impressions are condensed in the grey substance of the optic thalami, whence they irradiate towards the various regions of the cerebral cortex, and that in hallucinations there is a spontaneous activity of these ganglia with the like result, the cortical cells acting on the erroneous indications conveyed to them as if they were real. This theory, which is adopted by M. Christian, the distinguished head of Charenton, in the recently published French Encyclopædia of the Sciences,[1] does not appear to me to be satisfactory, for this reason among others, that it ignores that large class of cases which unquestionably originate in what we call mental, that is, cortical changes.

The difficulty of deciding whether, in hallucinations, the terminal sense-organs are involved, as well as the cerebral centres, confronts us also in the study of those hallucinations which are caused by the administration of drugs.

Dr. Lauder Brunton, who informs me that what follows correctly expresses his views, experimented on himself with a grain of digitalin taken in the course of forty-eight hours, and found that it produced distinct subjective sensations of sight.

As a similar effect is produced by the local application of

[1] 'Dict. Encyclopédie des Sciences Médicales,' 1885, an able article by Dr. Christian of Charenton.

this drug, it would be probably safe to conclude that the periphery, as well as the centre, is involved when digitalin is administered internally. The visions induced by Cannabis Indica are well known. Here, on the other hand, it is probable that the cerebral centre is the seat of these visual hallucinations, because the higher mental functions are so obviously under the influence of this drug.

Again, auditory hallucinations produced by the administration of drugs may be supposed to have their seat, in some instances, in the cerebral centre of hearing, and in others in the terminal apparatus. As strychnia has the effect of intensifying the sense of hearing by its action on the brain, it would be interesting to know whether those auditory hallucinations which arise in insanity are rendered more vivid by the administration of strychnia.

Lauder Brunton is himself by no means sure whether the singing in the ears caused by full doses of quinine or salicylate of sodium is due to its stimulating action on the auditory nerve or the cerebral auditory centre, although, seeing that delirium follows the administration of still larger doses, the probability is that this singing does not involve the auditory apparatus, but the cortical centre. The action of quinine upon the cerebral centres seems confirmed by a case recorded by Dr. Brunton, in which a dose of quinine was followed by an epileptic convulsion.[1]

Again, as some patients complain of bad smells of clearly cerebral origin, it would be worth while experimenting with strychnia, because it has been found to have the effect in healthy persons of neutralising the character of disagreeably smelling substances, such as assafœtida, and rendering them even agreeable. How strychnia acts may be uncertain, but Dr. L. Brunton is disposed to attribute its effect not only to the action which is produced on the cerebral olfactory centre, but possibly also upon the olfactory nerve itself, inasmuch as peripheral action has been demonstrated in the case of the retina of the frog by recent experiments.[2]

These facts, few though they be, lead me to hope that the

[1] 'Pharmacology, Therapeutics, and Materia Medica,' 3rd Ed., 1887, p. 190.

[2] This point is recorded and illustrated in Dr. Brunton's work, 'Op. cit.' p. 227.

further study of the influence of certain drugs upon the nervous system in inducing subjective sensory phenomena will throw some light upon the pathology of the hallucinations of the insane.

Do we gain any light from the occurrence of unilateral hallucinations? Are they induced by peripheral changes, or are they of central origin? Dr. Alexander Robertson, in a paper read at the Psychology Section of the International Congress, 1881, explains unilateral hallucinations by supposing that there is an original difference in the sensory centres of the cerebral cortex; one, in fact, being feebler than the other. He applies Sir James Paget's views on general pathology: namely, that "when a part is congenitally weaker than it should be, or has been enfeebled by disease, though it may be restored to an apparently healthy state, it is apt to be affected first, and to suffer most when a general morbid action arises in the system." Thus, alcohol might cause unilateral hallucinations, according to Dr. Robertson, by acting on the feebler cortical sense centre of one side.

On the other hand, M. Régis has endeavoured to show that unilateral hallucinations are an unanswerable proof of the intervention of the sense-organs themselves in hallucinations, the lesion in those hallucinations which are unilateral arising in the periphery, and being propagated gradually to the sensorium or cortical centre.

He adduces several interesting cases. In one, a patient heard voices applying gross epithets to him, in only the right ear. He laboured under purulent otorrhœa of this ear for a long period. In another case (reported by Pick of Prague) there were visual hallucinations restricted to the left eye, and also hemiopia. Here, ophthalmoscopic examination revealed defect of the visual field, limited to the upper half of the retina. Altogether five instances are given of unilateral hallucinations, manifestly caused by lesion of one of the senses on one side. In one instance the local treatment removed the hallucination. The inference is, therefore, drawn that unilateral hallucination recognises as a cause the unilateral lesion of one of the senses.[1]

[1] See 'L'Encéphale Mars.,' 1881.

The cases no doubt disprove the exclusive doctrine of Esquirol, that the sense-organs are in no wise concerned either primarily or secondarily with hallucinations; but they do not militate against Dr. Robertson's view, which would none the less apply to hallucinations not demonstrably of peripheral origin. Nor do M. Régis' cases touch the doctrine of Brewster, that centrally induced hallucinations must involve the sense-organs as well as the brain. I have already adduced evidence to show that Brewster's doctrine is not tenable in this exclusive way of putting it, any more than Esquirol's. No one can doubt that some hallucinations are of peripheral origin.

As an illustration of the uncertainty which surrounds this inquiry, and as a justification of introducing it for discussion, I may mention the fact that Dr. Alexander Robertson expresses himself very cautiously in regard to the sensory centres in the cortex in their relation to hallucinations. He observes: "I, to some extent, assume that the supreme centre in the cortical substance is the one involved. The fact that hallucinations are associated with other mental disturbances, and, indeed, in almost all the patients, with complete insanity, seems to point pretty distinctly to that as the correct conclusion. Still," he adds, "in certain cases disordered action may be limited to the sensorium." An interesting case in point is given, but Dr. Robertson makes the guarded commentary, "on the other hand, it is quite conceivable that the highest visual centres—the psycho-sensorial—may alone have been involved, just as the convulsive seizures limited to the arm or leg disease may apparently be confined to the particular centre in the convolutions which is believed to preside over the movements of the one or the other of these members."

In conclusion, I would submit the following propositions on the questions raised in this article:—

1. That lateral pressure on the eyeball, or looking through a multiplying lens, never doubles an object, unless it is actually external to the eye. That, therefore, either of these tests of whether an alleged object is really external or only a hallucination is trustworthy.

2. That the after-image of a luminous object, this being certainly on the retina, obscures or covers real objects, moves

with the motions of the eye, and is "projected" when the observer looks on a dark ground.

3. If visual hallucinations present the same phenomena as those observed in these luminous after-images, they may be regarded as involving the retina.

4. If, on the contrary, these phenomena are not present, visual hallucinations do not involve the retina, being confined to the cortical centres, or possibly extending to the sensorium, and may occur when the optic nerves are atrophied.

5. The discovery of cortical sensory centres appears to throw light upon cases of hallucination not of peripheral origin, whether their abnormal action be regarded as primary, or as secondary to that of higher cortical centres.

ON A CONNECTION BETWEEN EPILEPSY AND ERRORS OF OCULAR REFRACTION.

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SOME years ago, when conducting an investigation into the condition of the fundus oculi in insane individuals, we tested the refraction of a considerable number of Asylum patients: a fair proportion of these were epileptics; and in the course of this enquiry we were struck with the large number of refractive errors which we met with in the epileptic patients submitted to examination—a proportion considerably greater than that obtaining amongst the other classes of patients.

The number of cases then examined was not, however, sufficiently great to render it safe to draw any general conclusions, and we were unable at the time to pursue the subject further.

Since then, however, one of us has paid special attention to the matter in the case of patients with simple epilepsy coming under notice in private practice; and last winter we conjointly took an opportunity of examining upwards of one hundred Asylum epileptics, and testing their refraction with as much accuracy as possible. The result of the investigation of the matter on these lines has been to confirm our previous idea with reference to the large percentage of abnormalities of refraction amongst epileptics, and to make it appear probable to us that in certain cases the refractive error and the epilepsy may stand to each other in the relation of cause and effect.

To prevent any possible misconception, we wish at the outset to make clear that we have no notion of suggesting that all cases of epilepsy have their origin in ocular defects, but only that a certain proportion, at present undetermined, are thus produced. Nor indeed do we think that refractive errors, however great, would be capable of producing this affection in the case of individuals with thoroughly stable brains. But, given a predisposition to epilepsy, we believe that an abnormal condition of the refractive media of the eye may, at times, prove the exciting cause of the disease.

We are indeed now familiar with the fact that nervous troubles of other kinds have not unfrequently such a starting point. Headaches and neuralgias of different sorts frequently owe their origin to ocular troubles, and disappear when the patient is supplied with accurately adjusted glasses. There seems, therefore, nothing unreasonable in the idea that epilepsy may at times be similarly produced. Our aim in the present communication is simply to state what facts we possess in support of this assumption.

In our former enquiry we examined forty-eight cases of epilepsy associated with insanity, and we found that "out of the forty-eight cases, no fewer than twenty presented errors of refraction; of these twelve were hypermetropic, and two had hypermetropic astigmatism, two were myopic, and the other four had myopic astigmatism."[1]

Our present investigation may be divided into two parts. The first deals with a series of one hundred and three unselected cases of epilepsy associated with insanity, the patients being of both sexes and all of them inmates of Rainhill Asylum. The second division comprises nine cases of simple epilepsy which have come under notice in private practice.

We will consider the former class first. The cases coming under this head were taken altogether without selection, the object of the investigation being to determine the proportion per cent. of refractive errors in the class examined.

All the cases exhibited more or less mental defect associated with the epilepsy, some of the patients being imbeciles, whilst

[1] 'Brain,' Vol. VII. p. 29 (April 1884).

others had acquired their insanity later in life; but in every case, so far as known, the epilepsy had been the primary thing, and the mental trouble secondary. In all one hundred and three cases were examined, of which sixty-one were males, and forty-two females. Of these one hundred and three cases, we considered fifty-five to be emmetropic, including under this head such cases as did not manifest a higher degree of hypermetropia than one dioptre; whilst forty-eight cases (46·60 per cent.) presented refractive errors of a more or less decided character. These were proportioned as follows:—Hypermetropia accounted for eighteen cases, and hypermetropic astigmatism (simple or compound) for seventeen more; three were examples of simple myopia, and in seven others the myopia was associated with astigmatism; of the remaining three cases, two came under the head of mixed astigmatism, whilst the third presented myopic astigmatism in one eye, and hypermetropic astigmatism in the other (anisometropia).[1]

A few words are desirable as regards the mental condition of the patients. Of the one hundred and three cases, twenty-six were imbeciles, that is, the mental defect was either congenital or had come on in early life; in all of these cases in which a history was obtainable, the mental dwarfing appeared to have been the result of the epilepsy, and it is probable that this connection obtained in all instances. Most of the other seventy-seven cases presented more or less dementia, and in not a few this was very decided, the patients being perfectly fatuous; thirty-nine of the seventy-seven might be put down as decided dements. The remaining thirty-eight also exhibited as a rule more or less mental failure, their condition varying according to the frequency of their fits; in most of these, however, the symptoms of mania or melancholia (chiefly the former) largely predominated over those of mental failure, and in some few cases the patients in the intervals between their fits were in all respects rational.

The percentage of refractive errors above given seems to us considerably higher than that obtaining in the population at large; but this does not in itself prove that the fits were in

[1] We append a table showing the kind and amount of refractive error in each of these forty-eight cases.

any case dependent on the ocular defects. To establish this point, it would be necessary to show that the epilepsy disappeared when the abnormality of refraction was corrected with glasses. From the above record it will, however, readily be surmised that these patients were very unfavourable subjects for the application of any form of treatment. So readily does the nervous system fall into a habit, that if it has been accustomed to discharge itself irregularly for any length of time, this abnormal discharge will in most cases continue, even if the cause which originally excited it be withdrawn. To no disease is this statement more applicable than to epilepsy; and by the time this disease has produced sufficient mental failure to render it necessary to place a person under Asylum care, the case has usually lasted so long as to have passed altogether beyond the reach of cure. Nevertheless, we fitted up a few of the more promising of these patients with glasses, without, however, feeling in any way sanguine as to the effect of this method of treatment; and so far we have not obtained any decided results. The patients whose numbers are given as 2, 5, 13, 15, and 20 in the table, were thus supplied with correcting glasses. Of these, No. 20 died from an attack of status epilepticus not long after he had obtained his glasses, and before sufficient time had elapsed to test the result of this mode of treatment. Nos. 5 and 13 refused to wear their glasses. No. 2, who has suffered much from headaches, finds great relief from his glasses, and his first pair having been broken in a fight with a fellow-patient, he gave no peace until he was supplied with another one: his fits, however, appear to continue much as formerly; and the same remark applies to No. 15.[1] The results, as we shall see, in the case of private patients have been more satisfactory.

It may be objected to an enquiry on the above lines that ocular defects may be part and parcel of an abnormal physical constitution which expresses itself in the epileptic neurosis, and hence that we might expect to find a larger proportion of such defects in epileptic persons than in normally constituted individuals. But if this were the case, we should certainly

[1] The epilepsy in all these cases has been of many years' duration.

anticipate a higher proportion of refractive errors in imbeciles than in ordinary insane persons, since the former class comprise those who have either been congenitally defective, or who have broken down in early life—in whom therefore the hereditary vice has been greatest. But so far as our figures go the contrary is the case. Thus out of twenty-six epileptic imbeciles, we find fifteen to be emmetropic, the remaining eleven exhibiting some form of refractive abnormality—a percentage of errors of 42·30; whilst out of seventy-seven cases of epilepsy combined with other forms of insanity, thirty-seven exhibited abnormalities, giving a percentage of 48·05: so that a smaller proportion of refractive errors was found amongst the imbeciles than in the other classes of patients. The figures are not, however, sufficiently large to permit of decided conclusions being drawn.

In the second portion of our investigation are included all cases of simple epilepsy coming under notice in private practice. We would particularly call attention to the fact that none of these patients sought advice on account of any ocular troubles, but they were all sent to us by their medical attendants, to whom we had mentioned in conversation our ideas as to a possible connection between epilepsy and refractive abnormalities. It is noteworthy that of all the cases, nine in number, thus sent to us for examination, not one was emmetropic. We think it desirable to give a short epitome of every such case which has come under our notice.[1]

CASE I.—*Epilepsy.—Hypermetropia.—Cure.*

MISS J. D., æt. 11½ years, was brought on Oct. 6, 1887, with a note from her medical attendant, Dr. Henry Gorst of Huyton, stating that she had had frontal headache for some time, and had latterly had a series of convulsive seizures, which he considered to be of epileptic character. It was ascertained that the convulsive attacks, about forty in number, occurred on one day only, and on that occasion the patient was unconscious from about 9.30 A.M. until 10 P.M.

[1] Except when otherwise stated, no treatment other than the correction of the refractive error by glasses was resorted to after the case came before us.

Examination showed—

Right eye . .	Manifest hypermetropia	0·75 D = $\frac{20}{53}$
Left „ . . .	„ „	0·50 D = $\frac{20}{53}$

Oct. 10, 1887 (after atropisation for four days)—

Right eye . . .	Total hypermetropia	2·50 D = $\frac{20}{35}$
Left „ . . .	„ „	2·50 D = $\frac{20}{27}$

Spectacles of +1·50 D were ordered for near work, but were not insisted on for distance unless she had another fit. On May 2, 1888, the patient's mother wrote to say that the child "has had no more fits, and is free from the constant headache she suffered from. She now wears her glasses constantly." On further enquiry under date Aug. 21, 1888, the reply was, "She has had no return of the convulsions."

CASE II.—*Epilepsy.—Slight hypermetropia.—Ciliary Spasm.—Cure.*

MARY D., æt. 22; domestic servant. Sent by Dr. Morris. When 12 years of age was troubled with headaches and dimness of sight, and attended the Liverpool Eye and Ear Infirmary for a short time. Her first fit occurred in June 1885, and she had four on that day; for the next two or three months the attacks occurred daily, they then diminished in frequency to one weekly, and later on to one monthly until the beginning of January 1886.

Mar. 19, 1886.—*Ocular examination.*

Right eye	$\frac{20}{50}$ J 1 at 6 in.
Left „	$\frac{20}{50}$ J 1 „

Mar. 26 (after atropisation for a week)—

Right eye	Sph. + 1·25 D = $\frac{20}{50}$
Left „	Sph. + 1·25 D = $\frac{20}{20}$

Ordered + 0·50 D for near work. Atropine to be continued locally once daily for a fortnight.

Dec. 22, 1887.—No recurrence of fits. Can now read $\frac{20}{20}$ with each eye without her glasses; she always uses these for working.

Sept. 1, 1888.—Remains well. Has had no more fits.

CASE III.—*Epilepsy.—Hypermetropia.—Relieved.—Return of fits with discontinuance of glasses.*

BERTHA B., æt. 16; nurse; came under notice Dec. 30, 1886. The history was that four months previously she had commenced to suffer from pain in the centre of her forehead accompanied with a feeling of numbness in the nose. On Nov. 21, 1886, at 9 P.M. she fell down in a fit, and seven of these occurred between that time and 1 A.M. Nov. 22. The fits recurred on Nov. 26, when she had four, and again on Dec. 22, when she had three more.

Dec. 30.—*Ocular examination.*

Right eye . . . $\frac{20}{20}$ (perfectly) sph. + 1·75 D = $\frac{20}{20}$ p.
Left „ . . . $\frac{20}{20}$ „ sph. + 1·50 D = $\frac{20}{20}$ p.

Jan. 6, 1887 (after atropisation for seven days)—

Right eye . . . $\frac{20}{0}$ sph. + 4·50 D = $\frac{20}{20}$
Left „ . . . $\frac{20}{0}$ sph. + 4·25 D = $\frac{20}{20}$

Jan. 27.—Ordered glasses, right + 3·50 D, left + 3·25 D, for constant wear.
Feb. 20.—Both eyes $\frac{20}{20}$ (with glasses).
Mar. 14.—Patient returned to service.

From the date of being fitted with glasses, she wore them regularly up to the middle of May (1887), and during that time she had not a single fit. She then discontinued the constant use of the glasses, and only wore them occasionally; the result was, that at the end of May the fits recurred, and from that time until the end of the year she had them frequently and severely. Last February she gave up the use of the glasses altogether, and the fits continued to recur, at any rate until the end of May.

In this case the patient certainly seemed relieved by the glasses, since for 3½ months after commencing to wear them she had no fits; whilst these returned with the discontinuance of the glasses. The case illustrates the difficulty of getting young women to wear glasses when their sight is not obviously defective to themselves.

CASE IV.—*Epilepsy of fourteen years' duration.—Hypermetropic astigmatism.—No improvement.*

MARY ELLEN C., æt. 29; married; one child; has had fits since 15 years of age.

Jan. 20, 1888.—*Ocular examination.*

Right eye . . . $\frac{20}{100}$ (1 letter) $\frac{20}{70}$ (1) J 6
Left „ . . . $\frac{20}{40}$ (4 letters) $\frac{20}{30}$ (4) J 1

Jan. 23.

Right eye . . . Cyl. + 2 D (axis 60°) = $\frac{20}{20}$ (perfectly)
Left „ . . . $\frac{\text{Sph. + 0·25 D}}{\text{Cyl. + 1·25 D (axis 60°)}} = \frac{20}{25}$ „

Full correction ordered.

Aug. 23.—Patient has worn her glasses constantly since above date, but no improvement has resulted to the epilepsy, the fits continuing as frequently as before the case came under treatment. Since the epilepsy was of fourteen years' duration improvement was hardly to be expected.

CASE V.—*Epilepsy.—Compound hypermetropic astigmatism.—Cure.*

MISS CLARA P., æt. 21.

History.—At the age of 18 became a school teacher, but finding that evening study induced headache, she gave it up at the end of twelve months, and her headaches ceased simultaneously. After remaining at home for five months, during which time she was free from headache, she took the situation of a shop book-keeper, and her headaches recurred almost at once. On Aug. 23, 1887, whilst engaged in her ordinary work, she fell down unconscious in a fit, which her medical attendant (Dr. White of Warrington) states was an attack of epilepsy (*haut mal*). This was the first fit she ever had.

Sept. 17, 1887.—*Ocular examination.*

Right eye . . . $\frac{20}{70}$ (2 letters) $\frac{20}{50}$ (3) $\frac{20}{40}$ (2) J 1 (not well)
Left „ . . . $\frac{20}{30}$ (3 letters) $\frac{20}{20}$ (1) J 1 (fairly well)

Sept. 26 (after atropisation for a week)—

Right eye . . . $\frac{\text{Sph. + 3 D}}{\text{Cyl. + 1·50 D (axis 120°)}} = \frac{20}{20}$ (perfectly)
Left „ . . . $\frac{\text{Sph. + 2·25 D}}{\text{Cyl. + 1·25 D (axis 120°)}} = \frac{20}{20}$ „

Ordered glasses for constant wear.

Right eye . . . $\frac{\text{Sph. } + 1{\cdot}50 \text{ D}}{\text{Cyl. } + 1{\cdot}50 \text{ D (axis } 120^\circ)}$

Left „ . . . $\frac{\text{Sph. } + 0{\cdot}75 \text{ D}}{\text{Cyl. } + 1{\cdot}50 \text{ D (axis } 120^\circ)}$

On May 18, 1888 (eight months after being fitted with glasses) patient wrote to say that she had had no recurrence of the fit, her headaches were so slight as to be scarcely worth mentioning, and that she had never felt so well in all her life.

Sept. 1, 1888.—Remains well. No recurrence of fit.

CASE VI.—*Epilepsy.—Compound hypermetropic astigmatism.—*
—Relieved (?).

ISAAC P., æt. 14; sent by Dr. Bushby of Liverpool; has suffered from internal strabismus of left eye since the age of two. For the past 22 months has followed the occupation of an upholsterer. On Sept. 9, 1887, had a genuine epileptic fit; this was repeated on Nov. 20, and again on Nov. 30.

Nov. 29, 1887.—*Ocular examination.*

Right eye . . . . . $\frac{20}{35}$ (2 letters)
Left „ . . . . . $\frac{20}{9}$

Dec. 1 (after atropisation for two days)—

Right eye . . . $\frac{\text{Sph. } + 5{\cdot}50 \text{ D}}{\text{Cyl. } + 0{\cdot}50 \text{ D (axis vert.)}} = \frac{20}{35}$ (2 letters)

Left „ . . . $\frac{\text{Sph. } + 5{\cdot}50 \text{ D}}{\text{Cyl. } + 1 \text{ D (axis } 120^\circ)} = \frac{20}{9}$

Glasses ordered for constant wear.

Right eye . . . $\frac{\text{Sph. } + 4 \text{ D}}{\text{Cyl. } + 0{\cdot}50 \text{ D (axis vert.)}}$

Left „ . . . $\frac{\text{Sph. } + 4 \text{ D}}{\text{Cyl. } + 1 \text{ D (axis } 120^\circ)}$

Dec. 20 (with glasses)—

Right eye . . . . . $\frac{20}{30}$ (perfectly)
Left „ . . . . . $\frac{20}{200}$ J 16.

Dec. 21.—Resumed work.

1888. Had one fit on each of following days:—Jan. 22, April 5, May 12 and 19, June 17, July 7.

Aug. 2.—No fit since July 7.

Right eye . . . $\frac{20}{50}$ (perfectly)
Left „ . . . $\frac{20}{100}$ (1 letter) $\frac{20}{70}$ (1) J 8 (not well)

This case is still under observation, and it is yet too soon to pronounce a decided opinion upon it; but as the patient had three fits in seven weeks before being fitted with glasses, and these have since then diminished to six in eight months, there seems reason for thinking that some relief has been afforded.

CASE VII.—*Epilepsy.—Simple hypermetropic astigmatism.—No improvement.*

HENRY H., æt. 19; sent by Dr. Bushby.

On July 22, 1884, was three and a half hours in a swing-boat, and on the following day (July 23) had his first fit; had no more fits that year, but two occurred in the following year, two in 1886, two in 1887, and two in 1888 up to the time he was fitted with glasses.

Feb. 3, 1888.—*Ocular examination.*

Right eye . . . $\frac{20}{50}$ (5 letters) $\frac{20}{25}$ (1)
Left „ . . . $\frac{20}{50}$ (5 letters) $\frac{20}{25}$ (4)

Feb. 13 (after atropisation for a week)—

Right eye . . . Cyl. + 1·25 D (axis vert.) = $\frac{20}{15}$ (perfectly)
Left „ . . . Cyl. + 1·25 D (axis vert.) = $\frac{20}{15}$ „

Continued the use of atropine up to Feb. 19, when he began to wear his glasses. So far no improvement whatever has resulted therefrom; he has had numerous attacks of *petit mal*, alternating with decided fits, seizures of the former kind occurring pretty frequently, of the latter about once a month. Since the epilepsy in this case was of three and a half years' standing before the case came under notice, it could not be considered a favourable one for treatment.

CASE VIII.—*Epilepsy.—Myopia over-corrected with glasses.—Relieved.*

MR. ——, æt. 30: had a first epileptic fit when 18 years of age, and since that time he has had twelve such attacks, these occurring on an average about once a year; the last fit he had was on July 31, 1887, the day after visiting the Manchester Exhibition. He first wore glasses for myopia at the age of 16, and has since then twice increased the strength of the lenses. He came under notice on Aug. 19, 1887, and it was then found that he was wearing glasses two dioptres too strong.

Ocular examination—

Right eye . . . $\frac{20}{0}$ sph. $-3{\cdot}25$ D $= \frac{20}{35}$ (perfectly) $\frac{20}{25}$ (5)
„ $-3{\cdot}50$ D $= \frac{20}{25}$ „
Left „ . . . $\frac{20}{0}$ sph. $-4{\cdot}25$ D $= \frac{20}{30}$ „
„ $-4{\cdot}50$ D $= \frac{20}{20}$ „

Ordered, right, sph. $-3{\cdot}50$ D; left, sph. $-4{\cdot}50$ D. The glasses he had been wearing were, sph., $-5{\cdot}50$ D. each eye.

Aug. 26, 1888.—Since wearing the corrected glasses, patient has not had a single fit, that is an interval of upwards of a year has elapsed; so that we have ventured to put the case down as relieved; since, however, as long an interval has elapsed on former occasions, it is too soon to pronounce positively on the case. Patient has taken the bromides pretty regularly ever since his first attack, but less so of late than formerly.

CASE IX.—*Epilepsy.—Hypermetropic astigmatism.*

ELIZABETH R., æt. 19, came under the care of Dr. Davidson at the Liverpool Royal Infirmary, for epilepsy, to which she had been subject for two and a half years. There was a history of epilepsy in the family. The first fit, which occurred in November 1885, was immediately induced by a fall, in which her head was struck, and she had a series of fits, about forty in all, that day and the following; since that date they have recurred at pretty frequent intervals. The fits continued much as usual during the time she was under treatment at the Infirmary.

On Aug. 17, 1888, an ocular examination was made at Dr. Davidson's request.

Right eye . . . $\frac{20}{50}$ (4 letters) J 1 (fairly)
Left „ . . . $\frac{20}{50}$ (4 letters) J 1 (fairly)

Aug. 24 (after atropisation)—

Right eye . . . $\frac{\text{Sph. } + 0{\cdot}75 \text{ D}}{\text{Cyl. } + 1 \text{ D (axis vert.)}} = \frac{20}{20}$ (perfectly)

Left „ . . . $\frac{\text{Sph. } + 0{\cdot}75 \text{ D}}{\text{Cyl. } + 1{\cdot}25 \text{ D (axis vert.)}} = \frac{20}{20}$ „

Aug. 31.—Commenced to wear glasses.

Since this patient has only just begun the use of the glasses, it is manifestly too soon to give any opinion as to the result.

Briefly now to sum up the results of the foregoing cases. In two of these (Nos. 4 and 7) no improvement whatever has resulted; in each of these cases the epilepsy was of some years' duration, viz. 14 and 3½ years, respectively. Number 9 is of too recent date to permit of any conclusions being drawn with reference to it, and hence, in calculating results, it must be struck out. One case (No. 3) discontinued treatment, and hence we think it safer to eliminate it altogether, since, though relief was afforded in the first instance, it is manifestly impossible to assert that had the patient continued to wear the glasses, she would have remained free from fits. Two cases (Nos. 6 and 8) we look upon as relieved. The remaining three (Nos. 1, 2, and 5) we have ventured to put down as cured, no fits having occurred during periods varying from ten months to two years and five months. To No. 2, indeed, we think this term may fairly be applied; whilst in Nos. 1 and 5 we must wait for a longer period to see whether the cure is permanent, since intervals of this duration between fits not unfrequently occur, especially at the first onset of the affection. Out of seven cases then in which the results are fairly definite, we have two patients unimproved, two relieved, and three cured, so far as present appearances go. These cases are of course far too few to permit of any definite conclusions being drawn; the results are suggestive, but in no way decisive. Taken, however, in connection with the large proportion of refractive errors in epileptic lunatics, which we think we have demonstrated, they seem to us to justify the assumption, that certain cases of epilepsy do occur in which the attack is induced by the undue strain put upon the muscular apparatus of the eye by reason of an abnormality of

refraction. This being so, if the refractive error is corrected with glasses, there is reason to anticipate that the fits will subside, provided that this treatment is resorted to at a sufficiently early period of the case.

Future researches will doubtless determine the correctness or the reverse of this conclusion.

Postscript.

Since the data for this paper were collected, and our notes put into shape, our attention has been called (by a review in the 'British Medical Journal,' May 5th, 1888) to a work recently published in America, by Dr. Stevens of New York,[1] which in part traverses the ground we have gone over. Into the general subject of functional nervous diseases we cannot of course now enter, but the section on epilepsy has much interest for us, inasmuch as we find that the author's researches confirm our own conclusions independently drawn. Dr. Stevens has indeed found a considerably larger proportion of refractive errors in epileptics than has come under our own cognisance, only eighteen out of one hundred consecutive cases being emmetropic. Several interesting cases are detailed, in which the correction of the refractive error by appropriate glasses was followed by the complete cessation of the epileptic fits, whilst in other cases in which the cure was not absolute, considerable amendment ensued. In some of the cases, however, tenotomy of one of the recti muscles, owing to insufficiency of its opponent, was practised in addition.

[1] 'Functional Nervous Diseases; their Causes and their Treatment,' by Geo. T. Stevens, M.D.

TABLE SHOWING THE KIND AND AMOUNT OF REFRACTIVE ERROR IN FORTY-EIGHT CASES OF EPILEPTIC INSANITY, IN WHICH THE REFRACTION WAS ABNORMAL.

No.	Initials.	Sex.	Age.	REFRACTION.
1	J. H.	M.	31	High hypermetropic astigmatism
2	J. S.	M.	31	Compound hypermetropic astigmatism— Right eye . . Sph. + 2·25 D / Cyl. + 0·50 D (axis vert.); Left „ . . . Sph. + 2·25 D / Cyl. + 0·50 D (axis vert.)
3	E. C.	M.	25	Right.—Slightly myopic Left.—Slight mixed astigmatism
4	T. C.	M.	34	Simple hypermetropic astigmatism
5	J. H.	M.	29	Compound myopic astigmatism— Right . . . Sph. − 10 D / Cyl. − 3 (axis 60°); Left . . . . Sph. − 12 D / Cyl. − 3 D (axis 45°)
6	T. D.	M.	30	Myopia.—Right − 2 D. Left − 2 D
7	T. D.	M.	51	Simple hypermetropic astigmatism— Right . . . Cyl. + 2·50 D; Left . . . . Cyl. + 2 D
8	P. C.	M.	42	Hypermetropia.—Each eye + 2 D
9	R. A.	M.	39	Simple myopic astigmatism
10	J. B.	M.	42	Right.—Slight hypermetropic astigmatism Left.—Emmetropic
11	R. B.	M.	21	Right.—Myopic astigmatism Left.—Myopia
12	J. B.	M.	52	Hypermetropia.—Each eye + 2 D
13	E. D.	F.	34	Compound hypermetropic astigmatism— Right . . Sph. + 3 D / Cyl. + 3·50 D (axis vert.); Left . . . Sph. + 3 D / Cyl. + 3 D (axis vert.)
14	S. H.	F.	28	Myopia.—Right − 3·50 D. Left − 2·50 D
15	F. A.	F.	17	Hypermetropic astigmatism— Right . . Sph. + 0·25 D / Cyl. + 1·25 D (axis vert.); Left . . . Sph. + 2 D / Cyl. + 1 D (axis vert.)
16	J. T.	M.	33	Simple hypermetropic astigmatism
17	G. S.	M.	31	Hypermetropia.—Right + 1 D. Left + 2 D
18	W. L.	M.	63	Hypermetropia.—Right + 2·50 D. Left + 2·50 D
19	H. K.	M.	17	Hypermetropia.—Each eye + 1·50 D
20	T. F.	M.	32	Hypermetropic astigmatism— Right . . Sph. + 0·50 D / Cyl. + 4·50 D (vert.); Left . . . Cyl. + 1·50 D (vert.)
21	T. T.	M.	35	Hypermetropia.—Each eye + 1·25 D

No.	Initials.	Sex.	Age.	Refraction.
22	H. P.	M.	35	Compound hypermetropic astigmatism— Right . . Sph. + 1·75 D / Cyl. + 0·75 D (axis horizontal) Left . . . Sph. + 1·75 D / Cyl. + 0·75 D (axis 30°)
23	E. P.	M.	44	Right.—Simple myopic astigmatism— Cyl. − 2·50 D (vert.) Left.—Compound myopic astigmatism— Sph. − 3 D / Cyl. − 5 D (axis vert.)
24	E. C.	M.	39	Hypermetropia.—Right + 2·25 D. Left + 2 D
25	E. G.	M.	29	Compound hypermetropic astigmatism— Each eye . . Sph. + 3 D / Cyl. + 1·50 D (axis vert.)
26	J. N.	M.	33	Hypermetropia.—Each eye + 1·25 D
27	J. J.	M.	17	Hypermetropia.—Each eye + 2 D
28	C. M.	F.	39	Right.—Simple myopic astigmatism. Left.—Emmetropic
29	M. S.	F.	30	Hypermetropic astigmatism— Right . . Sph. + 0·50 D / Cyl. + 1 D (axis horizontal) Left . . Sph. + 0·75 D / Cyl. + 0·75 D (axis horizontal)
30	J. P.	F.	37	Right.—Hypermetropic astigmatism— Sph. + 1 D / Cyl. + 0·75 D (axis 30°) Left.—Emmetropic
31	A. C.	F.	25	Compound hypermetropic astigmatism— Each eye . . Sph. + 2·50 D / Cyl. + 1·50 D (axis vert.)
32	A. M.	F.	9	Hypermetropia.—Each eye + 3 D
33	E. C.	F.	25	Hypermetropic astigmatism— Right . . Sph. + 1 D / Cyl. + 0·75 D (axis 120°) Left . . . Sph. + 1 D / Cyl. + 0·75 D (axis vert.)
34	F. K.	M.	35	Hypermetropia.—Each eye + 2 D
35	W. T.	M.	27	Hypermetropic astigmatism— Right . . Sph. + 1 D / Cyl. + 0·75 D (axis horizontal) Left . . . Sph. + 1 D / Cyl. + 0·50 D (axis horizontal)
36	P. B.	M.	19	Hypermetropia.—Each eye + 9 D
37	E. H.	M.	66	Simple hypermetropic astigmatism
38	A. S.	F.	25	Hypermetropia.—Each eye + 2 D
39	C. H.	F.	6	High hypermetropia
40	C. W.	F.	45	Simple hypermetropic astigmatism
41	S. W.	F.	46	Slight myopia

No.	Initials.	Sex.	Age.	Refraction.
42	E. W.	F.	50	Hypermetropia.—Right + 3·50 D. Left + 4·50 D
43	A. M.	F.	29	Hypermetropic astigmatism— Each eye. . $\frac{\text{Sph. + 1 D}}{\text{Cyl. + 1·75 D (axis vert.)}}$
44	E. W.	F.	23	Hypermetropia.—Each eye + 1·75 D
45	C. W.	F.	38	Compound myopic astigmatism— Right . . $\frac{\text{Sph. − 2 D}}{\text{Cyl. − 1·50 D (axis 120°)}}$ Left . . . $\frac{\text{Sph. − 3 D}}{\text{Cyl. − 1·50 D (axis horizontal)}}$
46	E. C.	F.	42	Right.—Simple hypermetropic astigmatism— Cyl. + 1·50 D (axis 120°) Left.—Simple myopic astigmatism— Cyl. − 1 D (axis horizontal)
47	N. B.	F.	28	Hypermetropia.—Right + 1·75 D. Left + 1·50 D
48	A. T.	M.	38	Simple myopic astigmatism— Right . . Cyl. − 4 D (axis vert.) Left . . . Cyl. − 2·50 D (axis vert.)

XEROSTOMIA (DRY MOUTH).

BY W. B. HADDEN, M.D. LOND., F.R.C.P.,

Assistant Physician to St. Thomas's Hospital, and to the Hospital for Sick Children.

ONLY a few examples of this affection have as yet been described. The subject was brought before the Neurological Society, and a patient exhibited in 1887 by Mr. Hutchinson. Next year an example came under my own notice, and the history of the case, together with Mr. Hutchinson's, are recorded in the Clinical Society's Transactions, vol. xxi.

The term "Xerostomia" (*ξηρός*, dry, *στόμα*, mouth) has recently been suggested for this condition by Mr. Hutchinson and myself independently.

A brief account of the disease, so far as its causation and symptoms are as yet known, may prove of interest. All the subjects were women, and most of them were between the ages of fifty and sixty-five; but in one the onset occurred at the age of twenty-three, in another at thirty-five, and in another at forty-four. The disease began suddenly in three cases, preceded in two by severe mental shock.

In all the cases the condition of the mouth is the same. The tongue is red, devoid of epithelium, cracked, and absolutely dry; its appearance is like that of raw beef. The inside of the cheeks, the hard and soft palate are also dry, and the mucous membrane smooth, shiny, and pale. The salivary glands appear normal, and no mechanical obstruction has been detected in their ducts. The general health remains unimpaired, and in spite of the absence of saliva, no digestive disturbance is noted by any of the observers. Articulation, however, is difficult, in consequence of the absence of moisture, and swallowing has to be assisted by constant sipping. Such are the symptoms which are common to all the cases. It must also be added that the

disease was neither preceded nor accompanied by any special nervous symptoms, with the exception of the nervous shock at the initial period in the two cases already alluded to.

There are some additional points which may or may not prove to be important subsequently. Some of these symptoms have simply been recorded in isolated cases, or have received attention from two or three observers only.

In two cases there was dryness or deficiency of moisture of the pharynx; in two the secretion from the nose was diminished; in two the lachrymal secretion was arrested; and in two the skin was dry. In two instances falling out of the teeth is recorded.

Taste seems to be natural when substances are in solution. In my own case common sensation of the inside of the mouth and on the tongue was unimpaired, and the reflex action of the palate was present.

The urine when examined has been found normal, and it is expressly stated in two cases that sugar was not present.

Of this affection Mr. Hutchinson appropriately remarks: "The condition appeared to me to be an exaggeration assuming permanency, of the state of mouth which is common under the sensation of fear, and during states of nervous excitement." This practically embodies the view which I briefly put forward in the Clin. Soc. Trans. vol. xxi., viz. that the suppression is not dependent on gross change in the glands, but should probably be ascribed to some disorder of the nervous apparatus. When my patient first came under observation, I made enquiries with the object of ascertaining if there was known to physiologists any one centre controlling the secretion of all the salivary and buccal glands. Though experimental observation is silent on this point, there can be no doubt that such a centre does exist. This view is put forward by Mr. Hutchinson in the 'Illustrated Medical News,' Jan. 26th, 1889, p. 76.

The disease reaches its greatest intensity rapidly, and sometimes suddenly, and then remains without change for years. Such seems to be the rule. In my own case the administration of jaborandi, in doses of thirty to forty minims three times daily, had a very beneficial effect, the mouth becoming moist about a quarter of an hour after each dose. I omitted the drug for

a time, but the patient soon returned, and asked me to give it her again. Dr. L. Morgan [1] has also noted great improvement under jaborandi. Pilocarpin, in my experience, is less satisfactory; and this again is in accordance with Dr. Morgan's experience. Mr. Butlin [2] found benefit from hydrochlorate of pilocarpin in doses of $\frac{1}{12}$ of a grain. It is right to mention that according to Mr. Hutchinson and others, jaborandi was administered without any success Mercury, quinine, steel and opium have been tried ineffectually. Dr. Broadbent has found iodide of potassium of some use. The application of glycerine to the mouth is of decided value. To my own patient I suggested a plentiful use of horseradish; but I could not satisfy myself that it was of service.

Note.—Since writing the above, I have come across an allusion to this condition of Dry Mouth in Dr. Weir Mitchell's 'Lectures on Diseases of the Nervous System' (Second Edition, p. 209). The patient, a woman of thirty-one, suffered from aggravated hysteria, such as retention of urine, stupor, paralysis, hyperæsthesia and aphonia. At one time she had anuria, and then "complete absence of saliva, so that for a time the mouth was absolutely dry."

[1] 'Medical Press and Circular,' January 2, 1889, p. 4.
[2] See Report of Clinical Society, 'Brit. Med. Journ.' Nov. 3, 1888.

THE EPILEPTIC PAROXYSM.

Supplementary Paragraph.[1]

BY JAMES OLIVER, M.D., F.R.S. (EDIN.).

Tongue-biting.—Evidence of tongue-biting, although of some worth as an aid in the diagnosis of epilepsy, is not, to my mind, an infallible manifestation—when present—of the disorder in question. On several occasions I have witnessed attacks in women in which the movements were typically hysteroid or purposive in character, and yet these patients, after many of the convulsive paroxysms, revealed a laceration of the tongue. It might, of course, be suggested, and plausibly too, that the tongue-biting in such cases was a mere casual coincidence. The fact, however, that its recurrence was frequent dispels, to a certain extent, the feasibility of mere accidental association. If there be any slight dissociation in the movements of the lower jaw and tongue in death, as occasionally happens in the act of mastication, the tongue may be, and frequently is, bitten. In epileptic and other convulsive attacks, tongue-biting is probably the result of lack of harmony in the movements of the tongue and lower jaw.

Evidence of laceration of the tongue is not so commonly noted as many authors would lead us to believe, and probably after all, when considered with the frequency of attacks, is a mere coincidence.

As far as my own observations go I am convinced that the tongue is always bitten on that side which is the seat of initial motor spasm; this side corresponds with that towards which the head and eyes are primarily directed. It is to be remem-

[1] [To be read in connection with the author's paper (see page 343 of the present volume), from which it was accidentally omitted.—Ed.]

bered, however, that the head is sometimes during the attack deviated—rapidly it may be—from side to side, or directed primarily to one side, and it may even, throughout the greater part of the actual attack, be deviated to the side away from that which evinced initially the motor spasm. In noting, therefore, the relationship of tongue-biting with deviation of the head and eyes, the attack must be witnessed from the beginning, as the head may, at any period of an epileptic attack, change its direction. Foot clonus, as I have elsewhere remarked, can often be elicited after an epileptic paroxysm, and I have invariably noted that this phenomenon is more marked, and more readily produced on the side corresponding with that on which the tongue is lacerated. Now foot clonus is more easily developed, and continues longer in existence on that side on which the convulsion was greater, and this is usually that first attacked.

Regarding the mechanism of tongue-biting in epilepsy, many suppositions have been entertained as explanatory. It has been alleged that the tongue, during the actual fit, becomes engorged because of an interference with the return of blood, and that the consequent increase in size of the organ is the cause of occasional laceration, the tongue thereby projecting between the teeth if the lower jaw should happen at any time during the attack to be depressed. If, however, tongue-biting during an epileptic seizure were due to mere engorgement and swelling of the organ, one would expect to find it bitten more frequently on both sides during the same attack. Lately I saw a patient in whom the muscles of the right half of the tongue had gradually atrophied, and during the early days of this slowly progressive change the tongue was frequently bitten on the right side during mastication. In this case the muscles of the left half of the tongue being inefficiently opposed by those of the right, thrust the organ between the teeth, and thereby laceration of the right half resulted. Adaptation to the structural change eventually ensued, and now, although the atrophy of the right half is most marked, the tongue seldom is bitten. Some authorities state that laceration of the tongue during the epileptic fit is due to the organ being protruded beyond the teeth, and thereby caught.

Protrusion of the tongue is a movement habitually associated with depression of the lower jaw, and consequent elevation of the hyoid bone. If, therefore, the protrusive theory be accepted, the organ can only be lacerated when the movements are dissociated. The fact, however, that the tongue is invariably bitten on that side on which the motor spasm is first manifested tends to thwart this view. If laceration occurred during the act of protrusion the organ would necessarily be bitten on the side away from that which was the seat of motor spasm. That the tongue is bitten by a process of retraction and dissociation of movements I have no misgivings.

Clinical Cases.

A CASE OF ATAXIC PARAPLEGIA, WITH AUTOPSY.

BY CHARLES L. DANA, A.M., M.D. (NEW YORK).

MOST cases of ataxic paraplegia are cases of locomotor ataxia with extension of the process; or cases of lateral sclerosis with extension of the process. These latter cases originate, as a rule, in a somewhat diffuse transverse myelitis affecting chiefly the two lateral columns, and from this point secondary degenerations extend.

There are sometimes cases of primary sclerosis of all the long-fibre columns of the cord.

Such are about the views expressed by me in a paper on "Spastic Ataxia"[1] read before the New York Academy of Medicine two years ago ('New York Medical Record,' July 2, 1887), and in a discussion in Dr. Putnam's paper on this subject a year ago.

The following case illustrates the pathology of one form of this affection very well.

The following is the outline of the history:

Male, age 50.—Syphilitic infection in 1863, acute and chronic lead poisoning, colic, left facial paralysis, amblyopia, iritis. Gradual onset of a spastic paraparesis, with later development of ataxia, until spastic, and ataxic, and paretic symptoms were about equally prominent. Sphincters not involved. Some anæsthesia, little pain.

Death from diarrhœa.

Autopsy.—Lateral sclerosis of entire length of cord, most marked in mid-cervical region; slight posterior sclerosis in cervical region; chronic lepto-meningitis.

Nerve roots and peripheral nerves normal.

[1] In my original paper on this disease ('Medical Record,' July 2, 1887) I used the name "spastic ataxia," because in all my cases, which number now over fifteen, I have found the chief symptoms to be spastic ataxia, while the paralysis was not marked, there being paraparesis only. Dr. Gowers, however, first published the description of the symptoms, and is entitled to christen his child, though my own article was written before his was published.

I adopt the name "ataxic paraplegia," therefore, in the present case.

Jos. M., U.S., 50, single, painter, was admitted to my service in Bellevue Hospital, May 1st, 1888. He gave the following history: His occupation was that of painter.

In 1863 he contracted syphilis, and had secondary symptoms.

In 1870 he had a sudden temporary left facial paralysis.

In 1875 he had an attack of rheumatism (or lead poisoning).

In 1882 he had an attack of lead colic lasting six weeks.

In 1883 he suddenly lost his sight while at work. He improved very much in two months.

In 1884 he had an attack of colic lasting one day.

In 1885 he was admitted to hospital suffering from lead colic. He also had paræsthesia of right side of face and right leg, and headache.

Physical examination at this time showed a feeble paraparetic gait, but no ataxia in arms or legs, no anæsthesia or analgesia. Slight blue line on gums. The deep reflexes were decidedly exaggerated, the superficial normal.

Electrical examination of the muscles of the thighs and legs showed that they reacted with abnormal readiness to the faradic current (with secondary coil, slide at 0). With the galvanic current the muscles reacted with from 12 to 16 Leclanché elements, the anode requiring from 2 to 6 more elements than kathode. (There was at the time no milleamperemeter in the hospital.) Nothing especial could be learned from the examination.

Patient was re-admitted to hospital October, 1886, and an operation for irido-choroideotomy performed.

Patient entered the hospital again in May, 1888, and then first came under my care.

His past history, as seen, was that of chronic lead poisoning, and nervous syphilis, and irido-choroiditis.

Physical examination at this time showed no marked lesions outside the nervous system and eyes.

Intelligence good. His gait was feeble and uncertain; but he could walk alone with care. His legs were weak, and decidedly paretic. There was marked ataxia, static and locomotor. The deep reflexes were greatly exaggerated, ankle clonus present, and cramps in the legs occurred.

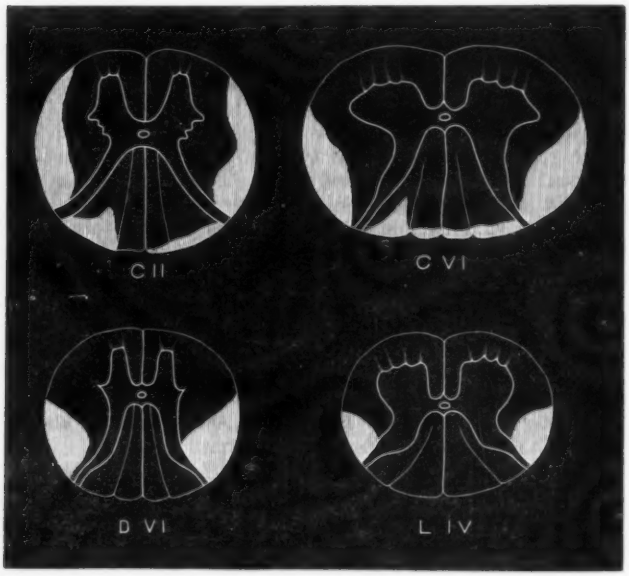
There was no special atrophy or paralysis of any muscle group.

There was a moderate degree of anæsthesia, tactile and pathic, in the legs below the knees, but not in the arms. The arms, however, showed ataxia even more than the legs.

Later in his disease he had a constant and severe pain in the right shoulder and down the back.

The bladder and rectal sphincters were normal. He had some pains in the back, and occasionally cramps and pains in the legs and shoulders.

He was shown to my class at the Post-graduate School as a very good example of ataxic paraplegia or combined sclerosis, the salient symptoms being—Paraparesis and arm weakness, aspastic condition of limbs, with exaggerated reflexes and ankle clonus; later, ataxia and anæsthesia, with occasionally



Diagrams showing sclerotic areas in the cord. Posterior leptomeningitic alterations not shown.

some pains in shoulders, and arm and back. No sphincter troubles.

The patient continued about the same until midsummer of 1888, when he died of an intercurrent diarrhœa.

Autopsy.—Body considerably emaciated. Thoracic organs not examined. Kidneys small and granular.

Brain.—Dura mater thickened in places, and adherent to brain over region of pericranian bodies. Cerebral arteries thickened and patent. No meningitis or other abnormalities.

Spinal cord.—Dura mater thickened, and some external

adhesive pachymeningitis is present in lower dorsal region of cord posteriorly.

On section evidence of lateral sclerosis and thickened pia is apparent.

Microscopical examination.—There is a chronic lepto-meningitis affecting the whole cord. It is not of a high grade, but is rather a slowly formed thickening.

Sections of the cord were made at various levels and stained in Weigert's hæmatoxylon, in aniline blue, congo red, and ordinary hæmatoxylon.

There was found a well-marked lateral sclerosis throughout the cord from the first cervical to the sacral segments.

The process was most marked in the mid-cervical region, where upon one side is evidence of nerve-tissue destruction and a sclerotic patch.

In the cervical region the posterior columns are affected in parts somewhat deeply, and throughout with a marginal sclerosis.

Below the cervical region the lesion is chiefly and essentially a descending lateral degeneration, the anterior columns not being at all affected.

Numerous examinations were made of the anterior and posterior cervical roots and of the anterior tibial nerve, but with negative results. The anterior tibial nerve simply shows some increase in its connective tissue framework, but no parenchymatous change.

The following is a more minute record of the microscopical examination :

1*st Cervical segment.*—Membrane decidedly thickened, and, where torn off, a very thin layer of medullary tissue comes with it. It is a little thicker posteriorly, and especially near the exit of posterior root.

Blood-vessel walls thickened.

Posterior roots show a little connective tissue increase, but no notable loss of fibres.

White matter.—Moderate degree of degeneration of lateral and one posterior columns as indicated in cut. Increase of interstitial substance, but no actual sclerotic focus. Vessels walls thick.

Gray matter.—Great increase of connective tissue cells around central canal. A good many enlarged vessels with thick walls.

Cells of anterior horn are apparently diminished in number and size.

Mid-cervical segment.—Meningitis as above, but on each side more deeply adherent to lateral columns, and at one side a

deep excavation is present where these membranes have been torn away. Anterior roots on each side here show no degeneration.

White columns.—On one side is an excavation produced by tearing away meninges, and here is found a marked sclerotic focus with degeneration of nerve fibres. A less marked degenerative area is seen on the other side.

The anterior columns are normal, including the antero-median.

The periphery of the posterior columns is decidedly affected, there being loss of nerve fibres without, however, any marked increase of connective tissue. It looks as if the change were due to imperfect nutrition from the inflamed meninges.

The grey matter.—The cells of the anterior horns are somewhat diminished in number. There is increase of connective tissue nuclei around the central canal and thickened blood-vessels.

The sclerotic and degenerative changes are decidedly more marked in the mid-cervical than at the first and second cervical segments, and the anterior columns were not at all affected. There can be no doubt, therefore, that the morbid changes were not secondary to cerebral lesions, although unfortunately the brain and medulla were spoiled and could not be examined.

In the dorsal and lumbar regions there is seen only the lepto-meningitis and lateral sclerosis. The gray matter and its cells are normal, including Clark's columns.

Sections of the anterior tibial nerve and of the anterior and posterior nerve roots showed no marked changes.

The pathological diagnosis is, therefore, lateral sclerosis, posterior marginal sclerosis in cervical region, chronic lepto-meningitis.

The clinical diagnosis, ataxic paraplegia.

CASE OF EPILEPSY, IN WHICH THERE ARE PERIODS OF AUTOMATISM OF A VERY WELL MARKED NATURE.

BY DR. GEO. H. SAVAGE, BETHLEM ROYAL HOSPITAL.

I SAW this patient first in 1886, and he had been occasionally under Dr. Hughlings Jackson for many years before this date. He was, when I first saw him, aged sixty-one, and married.

He had been a silversmith, and had recently retired from business.

There was no history of neurosis in his family, he was sober, had never been intemperate, nor had he had syphilis, there were no signs of injury to his head; he said he had had fits for thirty-four years. The history he gives of the first fit is as follows: that after a period of intense work and worry, he saw a hideous face start up before him, this vanished when he rested from his work, but on resuming it, the vision reappeared. After this followed a vision of a landscape, then he lost his senses for a time.

Ever since he has had fits; these occur chiefly by day and are without any cry, aura, or any hallucination, he sleeps as a rule after their occurrence, he has bitten his tongue once or twice severely, the fits are irregular in their intervals, but occur about once monthly. His speech at times has been affected after a fit, so that he was accused of drunkenness; he has no local pains, no loss of sight, or any special loss of mental power or of memory. The first mental disorder was change of feeling towards his wife; thus from being a very loving husband he began to suspect that she wanted to poison him; next he took a dislike to Dr. Jackson, saying that he was in league with his wife to poison him with digitalis. I formed the opinion that he was quite unfit to transact any business affairs at the time I saw him, and I gave him "bromidia," as he was sleepless. For two years his wife and daughter managed him at home, but his wife dying, he became hard to control, and during periods of unconsciousness, he signed cheques and gave away money without having any recollection of what he had done.

He was admitted into Bethlem Hospital on June 6, 1888, on

medical certificates, stating that he believed there was a conspiracy among the doctors and others to murder him; he said portions of his body were being burnt in half moons.

The true epileptic fits were now rare, but memory defective, and he was full of suspicion. He is said to have more attacks of petit mal, and simple losses of consciousness—several of these may occur in a day; he had shown a tendency to wander from home without reason or recollection.

The circulation and respiration were normal.

Pupils equal and re-acting, fundus oculi healthy, tongue steady, slightly furred, appetite good, bowels regular. Knee reflex normal, speech slightly blurred. He said he felt perfectly well, he was restless and unable to keep long at one thing. One fit occurring a week after admission was thus reported: he did not cry out, both sides appeared equally affected, he did not bite his tongue or pass fæces or water involuntarily; after a short sleep he became restless, wanting to get out of bed, he was confused in manner, and his speech was unintelligible, pupils dilated, with no reaction to light, no unilateral weakness, skin perspiring freely.

Up to June 22 he had daily attacks of the petit mal; on this date pot. bromidi gr. xv. ter die was given, and this controlled the fits, none being noticed till July 29, when I was present during the fit, and I submit the following details:—

As I was walking through the ward, Mr. A. beckoned to me through a glass door, and when I joined him he appeared to be slightly excited and flushed. He asked me if I would sit down and listen to him; I agreed, and he began to talk about his former illnesses and the very great kindness shown by Dr. Hughlings Jackson; he was slightly emotional as he went on to speak of his visits to the doctor, then, without any warning, he passed on to talk quite irrelevantly about his late wife and of his sexual relations. At this time he pointed towards his penis with his right index finger. I noticed that he then looked fixedly before him as if in abstract thought. At first I fancied this was done to suppress his emotion at the thought of his late wife.

Then suddenly his head was turned to the right, and he began vigorously picking his left thumb-nail with his right thumb and first finger; after he had done this for a minute he turned his head over to his left shoulder—away from me—and began to pick his right thumb-nail so vigorously that I had to interpose to prevent its evulsion, yet evidently he did not feel any pain.

He desisted from his picking, got up without any hesitation or stagger, and walked slowly about the room; he walked to a

window, which he opened and looked out of, then he turned round and watched the birds in an aviary, with apparent interest; every act had the appearance of design and care, he avoided obstacles and directed his steps easily and freely.

He walked to the door where he had first seen me and appeared to see some one, yet when I placed myself in his way he did not recognise me, but changed his direction to pass by me.

I touched him gently, but he took no notice, and when I spoke quietly also he did not react, but when I clapped him on the shoulder and spoke loudly at the same time, he answered as one waking from sleep. He then relapsed into his somnambulistic state and remained in it for ten minutes longer. In all he was nearly half an hour unconscious.

On my speaking to him after his return to consciousness he apologised for breaking off the conversation about Dr. Hughlings Jackson, and said he did not quite understand those lapses of consciousness, but that he thanked God he had now no fits. Altogether he has had some six or eight of similar though less well remarked attacks while in this hospital, and since July he has had one genuine epileptic fit.

In this case then we have a man who is subject to true epilepsy, and who has also very well marked periods of automatism, and who in some of these has squandered money, and in others for half an hour at a time he has performed highly complicated acts unconsciously.

The peculiar way in which the picking of first one thumb-nail and then the other, associated with the rotation of the head, confirms Dr. Jackson's belief that these automatic periods are generally, if not always, post-epileptic.

The medico-legal importance of such states cannot be over-estimated.

LOCOMOTOR ATAXIA OF AT LEAST FIVE YEARS' DURATION, WITH LARYNGEAL CRISES, AND CHARCOT'S JOINT-LESION, TERMINATING IN GENERAL PARALYSIS OF THE INSANE.

BY HENRY HANDFORD, M.D., M.R.C.P.,

Physician to the General Hospital, Nottingham.

LARGELY through the observations of Dr. Savage the association of locomotor ataxia with general paralysis of the insane has been established. It has been recently shown [1] that not only the cord, but also the sciatic and the peripheral nerves undergo degenerative changes in the vast majority of cases of general paralysis. A few cases evidently *commence* by a degeneration of the cord, or of the peripheral nerves, or of both; and some authorities consider that most do so. The majority sooner or later develop ataxic symptoms more or less; and in some the ataxia is well marked, while the mental alteration remains somewhat in abeyance. The occurrence of well-marked locomotor ataxia in the middle, or towards the latter part of a case of general paralysis, cannot be said to be altogether infrequent. But it is far from common for general paralysis to supervene upon a typical case of locomotor ataxia of several years' standing. The following is an example of this condition. Unfortunately no post-mortem examination was made, as the friends refused permission, notwithstanding the most strenuous endeavours to obtain their consent.

CASE I.—S. V., aged 28, married, a framework knitter, was admitted into the Nottingham General Hospital under my care in February 1886, complaining of difficulty of walking. His father was an insane epileptic, and died in an asylum. The patient used to bathe a good deal in the Trent, even in winter. No history of syphilis could be obtained. About 3½ years ago he began to experience difficulty in walking, and frequently used to fall down. His sight failed, and he had attacks of vomiting, and occasional loss of control over bladder and rectum. In 1883 he was an in-patient in this hospital, and was diagnosed to be suffering from locomotor ataxia. He then had retention of urine and required

[1] D'Abundo, 'Paralisi Generale Progressiva,' Napoli, 1888; and 'London Medical Recorder,' 1888.

the use of a catheter. A few days before his admission on the second occasion, he fell and twisted his right knee, but felt no pain. "He often falls and bruises himself very much, but never feels any pain, and only finds out the bruises from seeing the discoloration of the skin." When I saw him, the knee presented the typical appearance of Charcot's joint lesion, and it remained very much in the same condition till his death 1½ years later. There could be no reasonable doubt that the joint lesion was started by injury, though, as will be seen later on, there was abundant clinical evidence of peripheral neuritis. On attempting to walk, the movements of the legs were most disorderly. After much oscillation he could at length stand fairly steadily with the eyes open, but fell at once when they were closed. In the left leg the knee-jerk was quite absent: on the right it could not be tested on account of the swelling of the knee. There was no ankle-clonus. On examining the muscular sense, it was found that he could not place the legs in any desired position, either with the eyes open or closed; and with the eyes closed he could not tell in what position the legs had been placed for him. But on attaching weights to the legs, he could readily detect a difference of 1lb., and in the arms, differences of a few ounces were easily perceived. He could distinguish correctly between a test tube filled with hot water and one filled with cold, both when applied to the legs and the arms. He could not feel at all a light touch with the finger on the legs. The prick of a pin could sometimes be felt like a touch, but without any pain. In the arms no loss of sensation was detected, though he complained that feeling was impaired. About six months before his admission the second time, the arms began to be affected. His hands and fingers, he said, "went dead," and he lost power in them. He frequently dropped things, and in lifting a glass or a cup, always had to use both hands. The lips, tongue, and pharynx were affected. He spoke very thickly, the lower lip dropped somewhat, and the mouth was slightly drawn to the left. There was loss of sensation in the mouth, so that he could not feel whether any fluid he wished to drink was in his mouth or not, neither could he tell whether the cup was touching his lips. After a short time, by rubbing the lips, &c., the feeling returned sufficiently to enable him to drink. At the time of his admission there was no muscular atrophy. During his stay in the hospital the gastric crises were marked, but infrequent. There were occasional shooting-pains but no incontinence of urine or faeces. While I was examining his throat, on his opening his mouth widely and throwing his head back to enable me to see better, he had a curious epileptoid attack, affecting the muscles of the face, lips, pharynx and larynx. The breathing was interfered with, the face became turgid and dusky, and the whole appearance was alarming. This condition lasted about half a minute. He said similar attacks used to come on when he held his head back to be shaved, much to the alarm of the barber. Sometimes the attacks last several minutes, and he falls, if standing at the time. He loses consciousness, and

remembers nothing of what happens while he is in the attacks. When these epileptoid attacks could be avoided the vocal cords were seen very readily. During quiet respiration the left cord was further from the middle line than the right, and its border was concave, showing some paralysis of the inner fibres of the thyro-arytænoid muscle. Both cords moved during phonation, but the left less freely than the right. He had some difficulty in swallowing solid food, and had to drink with each mouthful to enable him to swallow it. The tongue was protruded to the left and the face was drawn to the left during articulation and during the epileptoid attacks. Taste was tested with quinine, salt and sugar. It was very deficient, but not absent. Common sensation in the tongue was much diminished. The power of smell was sluggish. There was partial ptosis of the left eye. Sight was imperfect. The pupils were unequal: the right was dilated to the size of a No. 10 catheter, and the left was about the size of a No. 6. They did not react to the strong light of a mirror, and it was doubtful if they moved at all with accommodation. The discs were grey, and the connective tissue stroma was too plainly visible. He slept badly and was low-spirited. He said that for ten months past he had occasionally, especially after reading or thinking, suddenly lost his memory and power of attention. "He wants to do or say something, but cannot tell what it is." If he had been reading, he did not know what he had been reading about, and could not understand what he continued to read, and had to stop. In about a quarter of an hour he was all right again. After a few weeks he left the hospital and went to live with his mother and a married sister, but he was so quarrelsome that the sister left, and he remained alone with his mother. He had some kind of fit or congestive attack, and shortly after this, began to grow excited and sleepless, and used to sit up all night. Though in receipt of parish relief he took a new house, began to drive about in cabs, and buy all sorts of things he could not pay for. He told his relations he was going to take them to a big house in the country, where he was going to build a lot of factories. He drove up to my house in a cab and said his mother had a lot of money, and had started a line of omnibuses. He himself had plenty of money, and he wanted to be a private patient. He was very anxious to have all his teeth out, as he thought he could eat better with an artificial set. He persisted in this desire for several months. His teeth were remarkably good and sound, and never showed that tendency to fall out, which is sometimes observed in cases of locomotor ataxia. Next day he drove up to the hospital and told them I had advised him to have his leg off, and to have a cork one instead, and he wanted that done. After this he was removed to the Borough Asylum, and for the further notes I am indebted to the kindness of Mr. Evan Powell. The notes say: "He was admitted on Sept. 15th, 1886, with unmistakable signs of general paralysis. He was emotional, and occasionally burst out into tears without apparent cause. He had total loss of sensation in both feet and legs up to the middle of the thighs

Sensation was also lost in both hands and arms, nearly up to the shoulders, where he could only just feel the prick of a pin. The mouth was drawn to the left: there was ptosis of the left eyelid, and tremor in the facial and lingual muscles. *Sept. 26th.*—Epileptiform fit. *Oct. 24th.*—Hallucinations of sight and hearing. Paralysis of the soft palate causes much difficulty of swallowing. *May 9th*, 1887.—Has delusions that people he cannot see are pulling at his arms and legs. He died on July 4th, after being in the asylum 10 months.

Shortly after this case, I was examining the larynx of an epileptic woman, and as soon as she opened wide her mouth and threw back her head, she had an epileptoid attack, much resembling the one described in the earlier part of this paper. There was marked strabismus, at first conjugate to the left, and then convergent; the tongue was moved rapidly from side to side, and respiration ceased, and she became partially unconscious. She attempted to vomit, and then slowly recovered. The whole attack lasted two or three minutes. Subsequent attempts to examine the throat produced slighter attacks, more of a cataleptic nature. Eventually, by making her breathe deeply and rapidly, a good view of the cords was obtained, and a paralysis of the left cord was found. This explained a condition of partial aphonia which had come on suddenly a few days previously. The paralysis passed off in a short time, and its cause was not discovered.

The phenomena in these two cases were very similar. They appear to have been epileptiform attacks, originating in irritation of the laryngeal nerves. Long ago it was shown experimentally in the lower animals that irritation of a sensory nerve could produce an epileptic seizure. It is said that cases which appear to commence as ataxy, and change to general paralysis, should be looked upon as examples of the co-existence of the two affections, and the dominance of the symptoms of one or the other. As this patient had well-marked ataxy four and a half years before his death, it is difficult to suppose he had general paralysis at that time, as the course of the disease in that case would have been very slow; whereas when once mental symptoms were developed, death followed within a year, indicating a rather rapid course. When first seen, a year and a half before his death, he had no mental alienation whatever, except some depression, which was not unnatural in his unfortunate condition. The only signs at all suggestive of general paralysis were the epileptoid attacks, the inequality of the pupils, the facial paralysis, and the thickness of speech. The first was in some measure accounted for by heredity, as his father was an insane epileptic. Inequality of

the pupils is occasionally found in locomotor ataxy. The thickness of speech was sufficiently accounted for by the paralysis, and the impaired sensation in the tongue and lips. The characteristic tremor of the lips and tongue was absent at this time. I should mention that he early had some mental depression, but that did not at first suggest to me the oncoming of general paralysis; for I have long been accustomed to associate some appreciable degree of mental change with the majority of cases of chronic cord degeneration. It is natural that a disabling disease, not necessarily associated with much or any pain, should lead to a despondent view of things. But the mental aspect of patients with cord-degenerations is very different from that of the most hopeless, and, perhaps, equally painless, but always cheerful, cases of phthisis; and equally different from the sad resignation of malignant disease. The mental aspect in cases of cord degenerations has seemed to me to tend towards melancholia, with discontent and quarrelsome dissatisfaction. Though tabes dorsalis is said to commence most frequently in the lumbar region of the cord, the early occurrence of ptosis and of atrophy of the disc, indicates an implication of the cranial nerves, or medulla and basal ganglia, or all three in the degenerative process. It is reasonable therefore to infer, that of the same, or similar, character to the system degeneration in the cord, there is a degradation of that perfect state of nutrition of the cerebrum as a whole (we do not know what portion, if any, is the organ of mind), which is necessary for "*mens sana in corpore sano*," though it may not proceed so far as to be readily demonstrable by the microscope.

A CASE OF CEREBRAL TUMOUR, APPARENTLY THE DIRECT RESULT OF A CRANIAL INJURY.

BY WILLIAM DUDLEY, M.B.,

Assistant Medical Officer, West Riding Asylum.

For the history of this case up to the date of the patient's leaving the National Hospital, I am indebted to Dr. Ferrier, who very kindly lent me his notes, and gave me permission to use them.

Frank H——p, formerly a clerk, was serving in the Cape Mounted Rifles. In July, 1886, he was assaulted by three natives, and immediately lost consciousness upon being struck on the head with a stick. He remained unconscious about twelve hours, and during that time was seen by a medical man, who sewed up the wound. On recovery of consciousness he felt very weak, which he attributes to having lost much blood; but he had no paralysis anywhere, and felt only very slight headache. Three days after, whilst reading a novel, he had his first fit, which came on suddenly, without warning, and rendered him at once unconscious. He was told that he jumped up, screamed, got out of bed and struggled. He was put back to bed, and thinks he remained unconscious about an hour.

After the first fit, he had no more the same day; but the next day he had seven or eight. These differed from the first in the fact that he had a warning. A feeling as of a galvanic shock began in the right shoulder, ran down the right side, up again and down the right arm, up the arm again and two or three times up and down the right side and leg, and then, reaching the right side of the neck and head, he lost consciousness. When unconscious he believes he was stiff in his limbs, but did not struggle or cry out.

For a week he had four or five fits daily of this character, and during this time he suffered from headache which seemed to radiate from a point about two inches above the left ear and round to the other side of the head. After the first week of fits,

the warning sensation used to begin in the right arm, but he cannot localise the place of beginning. He says the arm felt as though running away from him, and he would put out the other to grab it, but never could. The sensation would spread to the lip and tongue, but since the first week he has never felt it in the leg. He then lost consciousness, and the spasms, he was told, passed down the right side and were confined to it. He had these fits for about three months, about twelve a day, coming on more often in the daytime, but occasionally in his sleep. The night fits were more severe than the day ones, and he sometimes became unconscious without the warning sensation.

Just after fits, for a few minutes, he used to have thickness and difficulty in speech, but recovered again. He also had at this time a little weakness of the right arm, and used to limp a little. He had no vomiting at any time.

The original wound healed about two months after the accident.

In February, 1887, he underwent an operation (trephining). For a time he was much improved. He felt quite well. He walked without limping, and had no fits for six weeks. Then he had another fit similar to the last described, which was shortly followed by others. On June 1st he started for England. Just on starting he had eight fits, and then completely lost power of speech, but had no marked paralysis. He understood what people said, and knew what he wanted to say, but could not get it out. He could walk about shortly after, but was slow in recovering speech, which has gradually improved to the present time (July 1st, 1887). During the voyage he had a great many fits of the same character as before. He arrived in England on the 18th of June. On the 28th he had three fits, two being severe and one slight. Since starting home he has been slightly lame again, and he has been a little deaf lately.

On July 1st, 1887, patient was admitted into the National Hospital for the paralysed and epileptic.

On admission.—Has slight weakness of right leg. Does not raise the toes well from the ground. There is weakness of lower part of right side of face.

There are two minute vascular dilatations on the outer side of right optic disc. The retinal vessels with which they are connected are not visible. No evidence of past neuritis.

Senses of smell and taste do not seem normally acute. He calls bitters sour.

He says there is one spot always tender, and pain persists for hours after it has been pressed on. The spot is exactly in midline, seven inches from root of nose.

Six inches from root of nose and one inch to right of median line is a depressed cicatrix from the former operation. On the left side is a depression in the skull at the anterior inferior angle of the parietal bone. There is some tenderness over the left parietal.

July 6th.—At 11 A.M. complained of a "sensation." For about a minute nothing particular was noticed, but patient seemed unable to articulate, and sat up in a constrained attitude, turning slightly to the right. He then sat for half a minute intently watching his right arm, and had his left hand open and in position to catch hold of right. The right arm was then somewhat rigid. At this time he lost consciousness. Directly after this, face turned to right, and violent twitching began at right corner of mouth, and extended over that side of face. There was extreme conjugate deviation of eyes to right, the pupils at first being hidden behind external canthus; when seen they were dilated. While the face was twitched the limbs were quite flaccid at first, but shortly after tonic, spasm first attacked the right arm, which became pronated and turned in. Then the convulsion came on in the left arm and leg and the face was turned over to left, and violent clonic movements began on that side, and then the eyes deviated to the left. Clonic convulsions were then general.

When the convulsion first began, patient turned very livid; but during the premonitory sensation there was no apparent change.

The pupils quickly regained their normal size after the convulsion ceased, and the patient regained consciousness. There was ankle-clonus on both sides for a few seconds after the end of the fit. Knee-jerks not remarkably active. No marked loss of power on right side.

Patient had several fits similar to the last described. Sometimes he experienced a "sensation," without a convulsion.

Shortly after "sensations" or fits, there was distinct loss of tactile sensibility all down the right side, including the right side of face, and right half of tongue. He could taste bitters and sweets on the left side of tongue, not on the right. When some time had elapsed without a fit or "sensation," these symptoms could scarcely be detected.

On September 13th, patient left the hospital in opposition to the advice of Dr. Ferrier, who was contemplating an operation. He came to his brother in Wakefield, and almost at once went to Leeds Infirmary, in which he stayed a fortnight. Then he spent a fortnight with his brother. On October 15th he had three fits; on 16th, one.

According to his brother, he has always had excellent health, apart from the injury, and has always been temperate.

Neuroses in the family were denied.

Patient was admitted into the West Riding Asylum, Oct. 17th, 1887. He recognised his progressing mental failure, and was very anxious that an operation should be performed on him. The risks and the possible results had been previously explained to him.

Oct. 17th.—Patient complains by pantomime of frontal headache. When asked to write down what is wrong, he writes "head" pretty distinctly, then he makes some unintelligible marks, and after obvious effort fails to get any farther. He recognises his failure, and is pained by it. When "aches" is written for him he nods assent. He understands all that is said, and occasionally can give an answer of a single word; he makes many attempts to speak, but most of his words are unintelligible. It is observed that the sounds have a more or less distant resemblance to the words he wishes to speak. He explains sufficiently that he can read and understand printed characters, but he fails almost completely to articulate them. Generally the first word or two has some resemblance to the correct one; then there is a confused jumble of sounds in which no resemblance to the correct sounds can be distinguished. He names letters correctly sometimes, but more often fails. If the wrong name is suggested he knows that it is wrong. His attempts at reading are obviously attended by great effort. He names most ordinary objects pretty correctly, but sometimes fails. So far as can be tested, his memory, except for words, appears good.

He does not know when a fit is coming on, but he is able to explain, by pantomime, the features of the fits before unconsciousness supervenes. The right side of the mouth is first drawn down, then the right arm becomes rigid, strongly supinated in the extended position; after this the right leg becomes affected, being extended and rigid. A feeling gradually extends from the leg up the right side to the top of the head (sometimes being painful). At this stage of the description he allows his head to fall back, shuts his eyes, and signifies by a motion of the hand that he becomes unconscious. In some fits, he explains, the right hand is pronated, with the fingers and thumb approximated at the tips into the form of a cone. The hand is never clenched. Synchronously with the spasm of the hand, he sometimes sees a blue light (apparently with both eyes) which appears to move away from him. After a fit his sight is not good, but he can give no account which would lead one to suppose that there is any hemianopsia.

Always during the fit he hears a humming sound like a bee, which he localises near the right ear. He notices no perversion of taste or smell. After a fit his right arm and leg generally feel weaker than those of the opposite side, and the foot feels numb and dead.

Tongue points slightly to the right, the right side of face is distinctly flattened, and naso-labial fold less marked; this is most distinctly seen when he smiles. The mouth is drawn somewhat to the left. No ptosis, no paralysis of ocular muscles. Knee-jerks about equal, normal. Plantar reflex less marked on the right side.

A week after admission he was put upon bromide on account of the distressing headache, and the medicine was continued about three weeks. Great relief followed this treatment and he became more cheerful and brighter; otherwise there was practically no change in his symptoms.

Dec. 26th.—The first fit since admission occurred. He was sitting in an arm-chair. Spasm commenced simultaneously in right arm and leg. The left arm and hand were kept across the chest, there was slight movement in the fingers only of this hand. Eyes directed to the right. Tongue not bitten. The convulsion lasted only two or three seconds, and he recovered consciousness almost immediately, but was more stupid than usual for a time. Three-quarters of an hour afterwards the eyes turned to the right and patient lost the use of the right arm; it seemed to fall dead to his side, but he quickly regained power in it.

Dec. 27th.—Seems as bright as usual. Says "Good morning" distinctly; asked to name a key, says "No, don't know;" he names a hat; calls a knife a "key," but immediately recognises the mistake. He names a watch, but fails to name the chain; calls a button "but." He cannot name a ring, and does not recognise the name when told it repeatedly. These tests evidently cost him great effort. He often appears as if about to speak, and then checks himself suddenly. He often closes his eyes as people do when trying hard to think. Sometimes he shakes his head and smiles, recognising his failure; if his first effort fails he is seldom successful after.

Dec. 29th.—Yesterday patient began to be drowsy and took little heed when addressed. To-day he is almost comatose, and has taken very little food. The pupils are dilated and inactive.

Stertorous breathing set in, and he died on Dec. 30th. Patient did not vomit while in the asylum, and during the last month he seldom complained of headache.

Autopsy.—The skull cap is less capacious in front on the left

side, owing to a slight projection inwards of the left frontal bone. In the coronal suture $\frac{3}{4}$in. to the right of the median line is a circular opening the size of a shilling, the result of a trephine. The margins are smooth and slightly thickened. The aperture is closed externally by the pericranium and internally by the dura mater; the latter is morbidly adherent for some little distance around the opening, and here it projects pouch-like above the general level so as to fill up the aperture in the bone.

On removal of the dura, the brain protrudes and shows marked evidence of increased intra-cranial pressure, the convolutions being flattened. The anterior border of the left hemisphere is somewhat concave, partly from the corresponding convexity in the bone, and partly from an undue prominence at its outer part. The left hemisphere is larger than the right; length, left 8$\frac{1}{2}$in., right 7$\frac{3}{4}$in. Greatest width of orbital surface, left 3$\frac{1}{2}$in., right 2$\frac{1}{2}$in.; greatest width of hemispheres in temporo-sphenoidal regions, left 3$\frac{7}{8}$in., right 3$\frac{3}{8}$in.

No morbid appearance is observed in the right hemisphere beyond the evidence of general pressure, and the projection into the pouch of dura mater of a part normal in appearance, of the anterior portion of the ascending frontal.

In the left hemisphere the third frontal and the lower part of the ascending frontal convolutions are much increased in size and altered in contour. The latter measures from side to side 1$\frac{1}{4}$in., and in depth 1$\frac{3}{8}$in. The surface of both has a glistening, gelatiniform appearance, and the pia cannot be removed. The two posterior gyri of the insula also are much increased in bulk; they are pushed prominently outwards and backwards and feel very hard.

On section of the left hemisphere, a tumour is found occupying the posterior part of the frontal lobe. It is of a somewhat conoid shape with the base upwards, where it measures 2in. transversely and 1$\frac{1}{2}$in. from before back. Traced downwards, it passes with an inclination outwards through the external capsule into the operculum, and the two posterior gyri of the Island of Reil, and from these a hardness, gradually vanishing, is traceable into the temporo-sphenoidal lobe. To palpation the tumour seems circumscribed, but it is seen to diffuse itself into the brain matter around. The lenticular and caudate nuclei are thrust inwards upon the lateral ventricle, the cavity of which is thereby almost obliterated, but the posterior corner of the left, the third ventricle, and the right lateral are dilated. The internal capsule shows no naked-eye changes.

For the most part the tumour is of a greyish-white colour, but

marked by numerous small bloodvessels. In parts the vessels are numerous enough to give it a rusty red colour, other parts are darker, mottled, or speckled by minute extravasations. In the centre is a focus of softening, the size of a small pea, surrounded by pigmentation. It is easily sliced; in the opercular gyri and at the periphery softer than elsewhere, almost gelatiniform, and of a slaty purple tinge. The two posterior gyri of the insula, on the contrary, are very hard, almost cartilaginous in feel, and of a dead white colour. The grey cortex is almost invisible, being indicated only by a very faint boundary-line of darker shade.

Elsewhere nothing abnormal is found. Microscopically the tumour presents the characters of the soft glioma.

The case had special interest during life from the question arising of the possibility of affording relief by surgical interference. In a healthy man with a good family history, symptoms of irritation of a localised area of cortex followed closely upon a cranial injury, and were attended by slowly advancing symptoms of paralysis pointing to the same region. When the patient first came under accurate observation, he had slight weakness of the right side of face and right leg, he was subject to convulsive seizures, preceded by a warning sensation in the right hand; the seizures began by turning of face to the right, twitching of right corner of mouth, deviation of eyes to the right, and dilatation of pupils; afterwards, spasm attacked the right arm, and finally became general. Some time previously he had lost the power of speech, but now had partly regained it. All this pointed to the third frontal, the posterior part of second frontal, and the middle of the ascending frontal, of the left hemisphere, as being the probable primary site of the lesion.

The nature of the lesion was by no means clear; but the rapidity with which the symptoms followed the injury seemed to me to exclude the idea of a new growth. The sudden supervention of coma prevented an operation, which the symptoms appeared distinctly to call for, and the autopsy disclosed a condition which could not have been benefited by operation.

A CASE OF TABETIC ARTHROPATHY.

BY F. ST. JOHN BULLEN,

Pathologist to the West Riding Asylum.

I. M., æt. 42, pattern-maker, married, was admitted into the West Riding Asylum on the 30th of October, 1888.

Patient's mental symptoms dated from one month before admission. The facts certified regarding him were: "Excited and wild in manner, restless, attempted to injure both his wife and mother, destroyed his furniture, said that he had invented a pattern which would yield him £12,000."

No predisposing element appears in the family history. Patient is stated to have been intemperate.

Mental State.—When examined the day subsequent to his admission, appears free from excitement, coherent, and ready of reply. He denies that he has shown violence towards any one, and insists that there is nothing amiss with him, and that the relieving officer must have brought him to this place—a convalescent home for imbeciles—in order to get rid of him. His house is the cleanest and his children the finest in Leeds. He states that he has invented a pattern for a new dress material which will realise to him £12,000, and to its purchaser £300,000. He is not rich now, but will be shortly, when he has sold the pattern. Is now an abstainer, but was a heavy drinker when a young man.

Physical Condition.—Patient is spare and of small stature. Cranium well-formed, and frontal development good. There is exaggeration of both patellar reflexes and of the plantar also. Neither ankle-klonos nor tap-phenomenon are obtained. Percussion wave good, no wasting of limbs, no rigidity, contracture, tremor or choreiform movements of extremities. Four trials with the dynamometer gave the following—$\frac{27}{31} : \frac{24}{30} : \frac{24}{30}$: with swing $\frac{32}{32}$. Equilibrium is well maintained, locomotion steady and orderly, co-ordination generally good. There is some feebleness of the lumbar muscles.

Sensibility is good in answer to tests by tickling, pricking, and of temperature; referred sensations are always accurate. The eyes are placed rather widely apart. Irides greyish-blue, no adhesions of iris. Pupils pinhole $\frac{1 \cdot 5}{1 \cdot 7}$ mm.; are fixed to light, accommodatory movements are present in both, but restricted.

Consensual reflex absent in both eyes; reaction to cutaneous stimuli as well as that with volitional movements abolished. Ocular media clear. Visual acuity = $\frac{5 \cdot 5}{20}$ metres. Visual field extensive and equal in both eyes. Colour tests applied, he fails only with blue. Movements of eyeballs perfect in all planes. Hearing is acute and equal both sides. Smell and taste are alike acute and discriminative. The tongue is steady, free from fibrillar tremor, ataxia, divergence or hemi-atrophy. Articulation is good. Skin is free from any hophoneuroses.

There is considerable swelling of the left leg, without much tension, and without any sign of inflammation. No evidence of thrombosis or other obvious cause of the condition exists.

Progress of Case.—The mental symptoms were variable; at one time denying his previous statements regarding the wealth to be derived from his invention, at another he would discuss his plans concerning it, and intimate that he still expected to derive great pecuniary advantages from it. Being importunate and irrational on the subject of returning home, he made his escape over the wall a fortnight after admission, but was soon recaptured. He gradually, however, became less optimistic and restless, more industrious, and improved in bodily health. Was wont to become emotional when questioned; his face had a rather heavy, expressionless aspect, the lips, especially the upper, being held very stiffly, almost immobile, during speech; articulation, however, was distinct. The tongue was slightly tremulous when protruded, and there was marked clonos of the eyelids when patient attempted to keep them firmly closed. Occasionally he had attacks of vomiting, to all appearance independent of gastric irritation and unaccompanied by pain. He stated that for two years previous to his admission into the asylum, he experienced pains shooting down the anterior surface of both thighs, very stinging and severe, which used to rouse him from sleep. Four months after admission he was placed in bed on account of a painless, chronic-like effusion into his left knee-joint. He became at this time once again restless, troublesome and unreasonable, saying "his leg would get well if he was away from here," and threatening to escape on the first opportunity. Stated that he had sold his pattern; did not know how much for, his wife was managing the transaction; wanted three suits of clothes that afternoon; had discovered that he possessed a splendid tenor voice, and meant to make a lot of money by it. He favoured his listeners with a rendering of "My pretty Jane," in a voice akin to that of a street Punch. There was at this time considerable tremor of lips and voice during ordinary speech; the pitch was also rather high, but patient refused to allow any examination of his throat. Speech rather hesitating and slurred. A month subsequently to the first appearance of the effusion into the knee-joint, the latter was found to present the same characters, although in the interim he had been kept in bed. The swelling, on examination, was partly bony, partly the result of effusion into the synovial sac, not transgressing the limits of

the latter. Not the slightest pain, tenderness, redness, or sign of inflammatory action existed. There was free mobility, accompanied with very coarse grating, and to some slight extent lateral movement was allowed. Patient insisted on getting up and walking about, as the joint gave him no trouble, and such appeared to be the case, locomotion being unimpaired except by his holding the leg a little stiffly.

By desire he wrote out a description of the onset of the affection, an abstract of which gave the following: "He was running and had a fall, bruising the right knee, which, however, gave him no further trouble. Nor did he notice anything amiss with the left knee until two or three days after, when he found it swollen to nearly twice the size of its fellow, and fancied he must have put it out of joint. He had no pain whatever in connection with it, although he manipulated it as roughly as possible, but simply a feeling of weakness after resting the limb for some time, also after leaning his weight upon it, he felt the joint give way suddenly at times, this again painlessly. After a month he got the swelling down somewhat, as he thought, by rubbing it and driving the blood on its proper course, but even then it was much larger than the other knee."

The ocular symptoms remained unaltered throughout. An important change in the symptoms was found at this date to be the abolition of the patellar reflex on both sides.

The mental symptoms had improved; he acknowledged his previous excitement, had large ideas of his own abilities, but either had lost, or was too cunning to show, delusion. Was discharged, relieved, about six months from the date of admission.

I had the opportunity of seeing him some weeks afterwards, and noted the following:—

"No trace of patient's former exaltation remains. He is quite rational, shows no pride in his vocal powers, nor has he any hope of making his fortune by the sale of his pattern. The patellar reflexes are absent. His gait is slightly but perceptibly affected, the feet being wider apart and the heels planted more noisily than in a normal individual. It is, however, difficult to dissociate the peculiarity of gait from that contributed by the knee-swelling. Equilibrium is plainly disturbed; he is unable to stand with feet approximated and eyes closed without considerable oscillation. When made to walk with closed eyes, there is a manifest uncertainty in his progress, and on turning round, a notable instability of balance. He is able to localise the position of his members well, approximating the fingers of both hands, touching his nose, and recognising figures described in the air with his forefinger, with eyelids shut; denies any numb sensation in the soles of his feet; describes sudden profuse sweatings, preceded by a hot feeling, which have affected him of late,

both during day and night, but more especially the latter time.

"Has not had as yet any gastric pain, but once since his discharge a recurrence of the attacks of vomiting. There is now very well-marked fibrillar tremor of face and lips, and to a less extent of tongue. Clonos of eyelids extreme on attempts of the patient to keep them closed. The pupillary anomalies are as before. The knee preserves the same appearance; there is evident bone-grating and distinct lateral mobility, but still no pain."

A CASE OF AMNESIA WITH POST-MORTEM APPEARANCES.

BY F. ST. JOHN BULLEN,

Pathologist to the West Riding Asylum.

S. C., æt. 73. Admitted into the West Riding Asylum on the 29th of November, 1887.

History.—Patient has always been a steady well-conducted woman. Eighteen months ago, she had a fall down nine or ten steps, cutting her face, but not being stunned nor otherwise suffering at the time. Six months subsequent to the accident, paralysis commenced in the right foot and gradually extended upwards to the right arm. The condition of speech at that time not accurately determined. The palsy was never entirely recovered from, and a second stroke seizing her nine weeks previous to her admission rendered the hemiplegia still more complete. Since this latter seizure she is said to have developed delusions, to have become vicious, unmanageable, and depressed.

Family History.—No insanity, apoplexy, &c.

Mental Condition.—There is much dementia, and it is not easy to isolate the patient's amnesic symptoms for this reason. Her memory is very weak, and her answers shew a great inappreciation of facts relating to time and space; relevant as a rule to the enquiry, they are often inaccurate, and she drifts invariably, if allowed, from the subject in hand and becomes incoherent. Understanding more readily simple requirements, she protrudes her tongue when told, gives her hand, and squeezes with it as desired, but does not discriminate between right and left member.

There is no doubt that she hears quite well what is said to her, although she does not always grasp its meaning. Asked her name, replies either, "I cannot, sir, I do not know them," or gives some Christian and surname, always the same one, but never her own, nor can she be made to give the latter, even as a repetition. She states her age is 24 or 34, that she has been a widow eight years, and that her husband was æt. 28 when he died.

Left to herself she rambles on incoherently, omitting very persistently substantives, except those of very familiar use, but using freely verbs, adverbs, and prepositions. Often substitutes words for those wanting, mostly quite of different meaning, and often devoid of any significance. A third apoplectic seizure occurring a few days after her admission, she remarks, "All this right side, this broken side, is a broken mess." Requested to

present her right hand and squeeze with it, says, "I cannot make anything of this, I am very much pained, I am very ill." Again, utters such as the following, "I was very broad when you knew me first; they knew that I had no breach in the earth, then they go out and think that I am bleaching, and I could not understand that I was breaching."

Her speech is, however, by no means always so incoherent as this, and, especially on being aroused suddenly from sleep, talks sensibly and correctly enough for a short time, excepting for the substitution of other words or sounds for the nouns occurring. She is unable to repeat what is said to her; shewn articles whose use she appreciates, and told their names, says, "Yes, yes, I know," impatiently, but is quite unable to repeat the word or anything like it. She is aware of her incorrectness or failure of diction, and states annoyedly, "I cannot make anything of this." This extends, as before said, even to her inability to pronounce her own name. There is no very prominent defect of visual acuity, as apparent by such simple tests as are alone applicable to her reduced mental condition. She recognises common objects, as indicated by the use to which she puts them, although in the majority of instances she is not able to assign to them their names. Asked the colour of a scarlet rug, replies, "Yes, I see, but I cannot sound it with my teeth." She has been able both to read and write, but there is now nearly absolute alexia and agraphia. Given a book of moderately large type, she is unable to pronounce words, even monosyllabic. Pointed out a single letter she can identify others, with fair precision when occurring in proximity, but if many words intervene she misses the letter when next met with. Does not mistake one symbol for another, as a rule, unless bearing some similarity of form, as, R, B, P. Does not recognise her own name when written out and presented to her. Asked to pick out certain letters, without a specimen being pointed out to her, she rarely, perhaps only by chance, chooses the correct one. Shewn a watch at twenty-five minutes to eleven, says, "Twenty-five;" on other occasions gives the hour or the odd minutes correctly; not seldom mistakes eleven for twelve, and becomes hopelessly lost when the dial is held upside down. Can read, generally correctly, the numerals indicating the day of the month at the top of a newspaper, though not the day itself, nor the month; if, however, a succession of figures is placed before her, she becomes quite confused over them, but can identify the first and last. She is unable to write by reason of the hemiplegic condition, and expresses her inability when a pencil and note-book are handed to her, by saying, "I cannot; I could, but this side is all wrong now."

Physical Condition.—Patient's expression is placid, and her countenance but sparsely wrinkled. She is able to frown a little; the articulation is somewhat impaired by absence of teeth and dryness of tongue. Pupils dilated, equal and mobile. There is neither facial nor lingual paralysis. Manifest weakness of the right upper extremity exists; myotatic irritability is a little increased; feeble coarse motility is possible—no contracture.

Patient only possesses little control over the right lower limb; the foot is maintained in a position of partial equino-varus, but not rigidly so; no ankle-klonos. Plantar reflex absent on right side, present on the left; testing of patellar reflexes not satisfactory. Locomotion is feeble and most unsteady; the right limb especially is dragged in walking. Owing to the patient's feeble intelligence, only coarse tests of sensation were applicable, but there was certainly some anæsthesia in the right lower extremity, doubtful in the corresponding upper.

Considerable rigidity of the superficial arteries; the urine contained sugar, no albumen. Throughout patient's habits were cleanly, excepting only the comatose stage preceding the fatal issue, and her behaviour quiet. She died seventeen weeks after admission; almost constantly purged and in a condition of semi-stupor for many days previous to death. Two evenings before the latter occurrence her temperature rose suddenly to 105·8°, falling the next day to normal.

Autopsy.

Brain.—Some chronic thickening of the inner membranes with considerable serosity is present, compensatory to more or less general wasting of the cerebrum. Previous to stripping the organ maintains a fairly plump, firm aspect; the arteries at the base shew numerous patches of atheroma, the middle cerebrals being especially diseased; their lumen is in several parts nearly occluded; on both sides they are distributed to the occipital region. There is recent blood film over the vertices of the left central gyri; the median end of the left ascending parietal is, above all other convolutions, wasted; the ascending frontal of the same side much less so, and not so much as the central gyri of the right hemisphere. The following is to be observed in the left hemisphere:—

There is great wasting of the posterior half of the superior temporo-sphenoidal gyrus, the portion anterior to this being also, though to a less extent, atrophied. The posterior third of the middle temporo-sphenoidal convolution, its continuation into the angular gyrus, and the latter itself, are converted to riband-like tenuity. Yellow softening affects the hinder part of the floor of the Sylvian fissure. The temporal annectant gyrus of the insular is still to be seen, but greatly wasted and softened, whilst the digitation anterior to it is perceptible only as a yellow-stained, slightly-marked ridge; the third and fourth digitations are sunken and widely differing from the plump, bold contour of those of the right insula.

The gyri constituting the operculum are apparently free from wasting.

In the right hemisphere the following parts were involved in yellow softening:—

The middle portion of the second temporo-sphenoidal; also an area the size of a crown-piece, which comprised the ensuing:

Posterior part of angular, second and, in part, third annectant gyri, second occipital and a portion of third, extending to nearly the tip of the occipital lobe. The posterior extremities of the first temporo-sphenoidal and supra-marginal also received extensions of the ramollissement to a slight degree.

On transverse sections of the hemispheres being made, it is found that on the left side, the softening of the gyri extends the whole of their depth as a pulpy mass, whilst a greatly diminished consistence of the white matter of the centrum ovale existed to nearly the posterior horn of the ventricle. Before the latter was opened, the medullary stratum forming its roof was seen to be free from softening, but of a reddish hue from the presence of a number of vessels. The ependyma of the ventricle had a hard, firm feel, and the white matter immediately surrounding the posterior cornu was of remarkable toughness, evidently sclerosed, and forming an effectual barrier between the softened and non-softened parts as a tough leathery wall, standing ridge-like above the neighbouring brain substance of lessened consistence. Beyond this condition of the walls of the cornu, nothing else is noticeable. At the level of section of the upper planes of the caudate nucleus, appear in the white substance two holes, severally the size of a small pea and a large pin's-head; these are the cut orifices of obliquely-placed channellings, opposite at this level, the anterior end of the posterior cornu of the ventricle: the larger opening is external of the two; tracing it towards the base it pursues a course having the direction, backwards, inwards and downwards towards the cornu; following it in the reverse direction, it makes its appearance externally in the lower part of the Rolandic fissure between the two central gyri. The smaller canal, situated internally to the preceding, opens by a small aperture in the cortical substance just anterior to it; traced backwards, it runs a somewhat parallel course and ends sooner in a cœcal excavation. The walls of these channellings are free from discoloration, and the surrounding white matter is but slightly below the consistence of the general medullary mass.

At the summit of the ascending frontal gyrus in the same section-plane is a small patch of softening, involving the deeper layers of the cortex and the subjacent white substance to an extent equal to the sphere of a small pea.

On slicing more deeply, so as to expose the segments of the lenticular nucleus, the capsules, thalamus, &c., it is obvious that the greater part of the pulvinar has undergone softening, so that its texture, though not actually broken down, is gelatiniform and tremulous. The geniculate bodies would appear to have escaped involvement, and the corpora quadrigemina are distinctly free from appreciable lesion, nor is there any perceptible change in the optic tracts. On the other hand, the pineal gland is much wasted; it remains as an irregular nodule, the size of a large pin's head; the peduncles are barely traceable. The posterior part of the outer segment of the lenticular nucleus, the external capsule, claustrum and cortical grey matter are affected by pulpy softening.

As regards the cortex corresponding to the insula, it is practically absent through the posterior half of the latter; further forwards it gradually passes from the linear to the stratified condition. The softening involving the third segment of the lenticular nucleus and the external capsule can be traced downwards, inwards and forwards, gradually diminishing in area, until passing beneath the anterior commissure, it ends in the grey matter of the basal surface.

Though it is questionable whether the posterior portion of the hinder division of the inner capsule can have altogether escaped involvement, yet the anterior bend is undeniably perfectly intact.

In the right hemisphere, very similar appearances as regards inward extension and distribution of softening exist, although the latter commences more posteriorly. From the cortex of the occipital lobe, inwards, the centrum ovale is involved in yellow softening until only a like leathery barrier to that on the left side separates it from the ventricle. In both cases the optic striations are completely severed, but on the right side neither optic thalamus nor insula have undergone change. The operculum like its fellow has also escaped.

There are three small cavities in the third segment of the R. lenticular nucleus and one in the claustrum, its posterior part, slightly involving the surrounding external capsule. From none of these does any tract of softening proceed. The outer third of the pes of left crus shewed degeneration.

NEURITIS OF THE MEDIAN NERVE.

BY WILLIAM L. RUXTON, M.B.,

Assistant Medical Officer, South Yorkshire Asylum, Sheffield.

SEVERAL points of interest, relating especially to the area of distribution of anæsthesia in lesions of the median nerve and the order of return of the various temporarily abolished lesions of sensation are brought out in the following case :—

T. P., on December 10th, 1886, fell heavily on a broken wash-hand basin, inflicting a wound on the front of his right wrist. The wound extended from the ulnar side of the radial artery to just beyond the middle line of the wrist. The tendon of the palmaris longus was divided, but careful examination failed to detect either division of the median nerve or any anæsthesia in its area of distribution. Under appropriate treatment the wound progressed favourably until the fifth or sixth day, when inflammation with slight suppuration in the depth of the wound set in, coincident with the appearance of which pain, described as shooting down the inner side of the thumb and radial side of the index finger, came on, was severe and was always worse at night. A spot exquisitely tender on pressure was also present, at the site of the original wound. The shooting pain gradually became less marked, and at this time the following condition was found :—Complete loss of sensation to touch, pain and temperature in the palm of the hand from rather behind the centre of the metacarpal bone of the thumb inwards as far as a line corresponding to the middle line of the middle finger, and forward as shown in diagrams A and B (p. 520) on to the thumb in front and slightly to the inner side but not extending to the tip, in the index finger involving the radial side only as far as the middle line in front, but scarcely so far behind, and in the middle finger also confined to the radial half. At all parts the anæsthetic faded gradually into the healthy regions as indicated by light shading in the diagram. Gradually the muscles of the ball of the thumb wasted so that the thumb could not be brought over the palm, and none of them ultimately reacted to either galvanism or faradism. The hand was easily affected by cold, and the skin became smooth and glossy, but otherwise healthy. He could not make a good fist.

Progress.—Limitation of the original area of anæsthesia was the first apparent change. Three months after the accident my notes show the following condition, when diagrams C and D were also taken.

Fig. A.

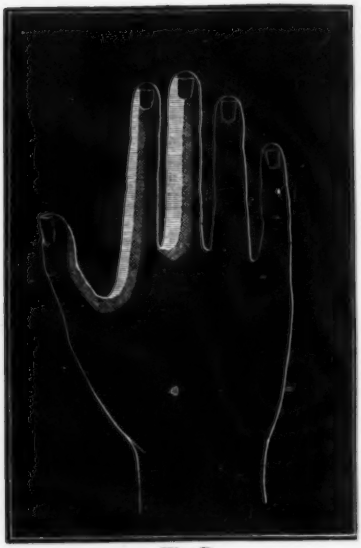


Fig. B.

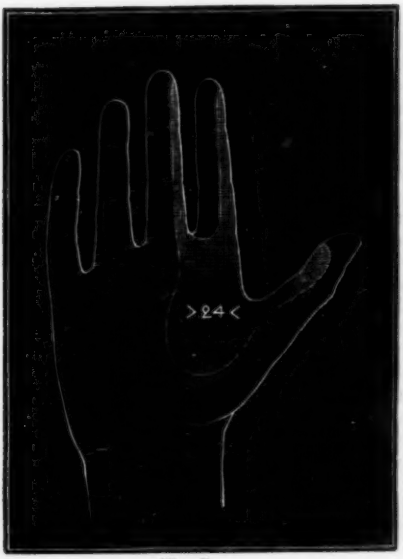


Fig. C

Fig. D.

1. Limitation of the original anæsthetic area. 2. Return of the appreciation of the sense of contact over all the primarily affected parts, though this sense is imperfect, everything being described as "woolley" or "gloved," and points being distinguished as two only in the palm at 24 mm. apart. 3. Return of the appreciation of pain over that area in diagram C indicated by lines in contradistinction to squares, and lastly there is as yet no return of the temperature sense in any part. By this time tumification of the wound had disappeared, and the painful spot was difficult to detect, perhaps absent. So far no voluntary movement of the paralysed muscles had returned. The next note, seven days later,

Fig. E.

showed that the temperature sense had commenced to return, and that variations of temperature could now be appreciated over the area shaded in diagram E, which shading is left open anteriorly to show that the return progressed from behind forward. The faradic current now brought the short muscles of the thumb into action, and voluntary power commenced to return, so that by the end of April he could bring the thumb across the palm and form a fairly good fist. The farther progress of the case simply showed gradual and ultimately complete return of sensation and motion, so that nine months after the accident, the hand could be freely used even for playing the piano or banjo.

The treatment adopted throughout was the frequent application

of galvanism, and an occasional counter irritant at the site of the injury.

Remarks.—The fact that no injury to or anæsthesia in the area of distribution of the median nerve was found immediately after the injury, that coincident with the formation of pus in the wound shooting pain aggravated at night, lasting some time, and paralysis and anæsthesia ultimately supervening, along with the fact that there was an exquisitely tender spot at the site of the original wound, proved clearly that the median nerve was not cut, and that neuritis supervened, ultimately resulting in destruction of the nerve's conductility. Three points are worthy of note. The anæsthetic area was peculiarly distributed; the anæsthesia was absolute, and the order of the returning senses was distinct and definite. With regard to the distribution, Ross[1] concludes that pathological lesions of the median nerve do not corroborate the description of the area of distribution as given in English text-books, but that the description given by Krause and Henle is more in accord; but this case very materially differs from Krause's description, especially, though not by any means entirely, in the fact that the ulnar side of the index finger was not implicated. That the loss of sensation to touch, pain and temperature was absolute, was verified time after time, the patient being under almost daily observation for months. Lastly, improvement followed a distinct and definite course; first, there was limitation of the original area of anæsthesia before any of the senses commenced to return; then came return of the sense of contact, next the appreciation of painful impressions, and one week later the temperature sense returned. In at least the two latter, improvement took place from behind forward, that is advanced from the lesion. The return of the appreciation of simple contact before that of painful impressions seems curious, but probably finds its explanation in the words of Bowlby,[2] that "a more intense impression is necessary to give rise to pain, and that a proportionately perfect nerve supply is requisite, while the sensation of touch can be more or less clearly perceived even when the nerve paths are more or less damaged." It may be asked, was not the nerve only partially destroyed? Evidently it was not even partially divided at the time of accident, and it is difficult to imagine that an acute neuritis could pick out only some bundles of nerve fibre; and even if this was so, the destroyed fibres would not be wholly distributed to the areas found anæsthetic.

[1] 'Brain,' vol. vii., p. 76.

[2] 'Lancet,' June 4, 1887.

A CASE OF TUMOUR OF THE BRAIN WITH A LONG HISTORY AND WITH FEW SYMPTOMS.[1]

BY W. B. HADDEN, M.D., F.R.C.P.,

Assistant Physician to St. Thomas's Hospital, and to the Hospital for Sick Children.

THE patient was the son of one of the porters at St. Thomas's Hospital. There was no history of epilepsy or other disease of the nervous system in the family. In 1875, being then eleven years of age, he had a bad fall whilst playing football. He received a severe wound above the right eye, and had to be carried back to the Hospital. I could not ascertain whether there was a fracture or whether he lost consciousness. He had occasional headache and irritability subsequent to this, but no further symptoms until December 31st, 1877, two years after the accident, when he had an epileptic fit. From this time until his death on April 15th, 1887, he had occasional epileptic seizures. The attacks usually occurred in series of four, five, or six, and the intervals varied from one to twelve months. According to the father, the convulsion began with turning of the head to the right, though it was always general. After the fit he "would wander round and round the room as if lost." He had also seizures at night, and then used to pass his water into the bed. He never suffered from attacks of *petit mal*.

Apart from his epilepsy the young man seemed quite healthy. At the time of his death he was a clerk in the steward's office at St. Thomas's Hospital. He was well-educated and good at arithmetic. He was a first-rate draughtsman, and if he had lived a few days longer would have entered for the examination of second-grade perspective and geometry at South Kensington. It was expected that he would have acquitted himself well, as I was informed by a gentleman qualified to speak on the matter.

The patient was a reserved and somewhat opinionated

[1] The young man had been an in-patient under Dr. Harley on two occasions, and under Dr. Bristowe once; and to these gentlemen, as well as to Dr. Sharkey, who had treated him for several years, I have to express my thanks for permission to describe the case.

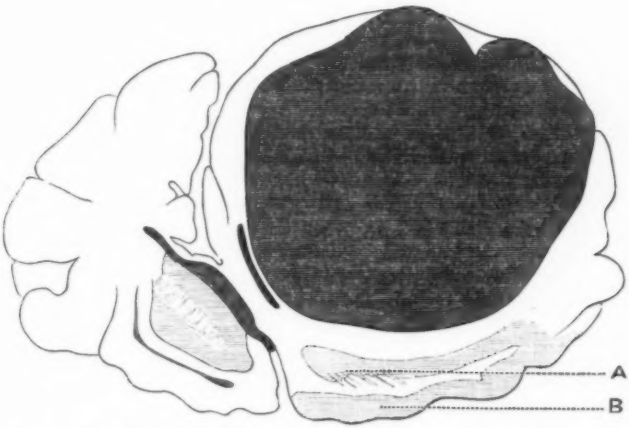
young man, but there were no signs of moral perversion in his character. From time to time he was treated by Dr. Sharkey, and subsequently by Dr. Harley, and I am informed by them that the fits were certainly controlled by bromide of potassium.

On February 7th, 1881, he was admitted under Dr. Harley, as he had had six fits in an hour and a half the previous day. For a whole year before that he had been free from attacks.

On June 20th, 1884, he was again admitted into the hospital, suffering from mental aberration after a number of fits which had occurred during the preceding two days. He was vacant in appearance, and would not answer questions. He often picked at the bed-clothes, but was not destructive. Once he got out of bed and ran about the ward. For two or three days a porter was required to watch him. In the state on admission it was noted that the pupils were equal and active to light and during efforts of accommodation; that the tongue was protruded straight; that there was no paralysis; and that the knee-jerks were brisk, but that there was no clonus. There was said to be general hyperæsthesia, with exaggeration of the superficial reflexes. In ten days he was discharged well, so far as his mental condition was concerned.

He was admitted for the third and last time on April 13th, 1887, unconscious and breathing stertorously. The house-physician's description was to the following effect. His face was pallid, and he was sweating freely. The urine was passed into the bed. The pupils were rather small and equal; they did not react to light. The corneal reflex was present, but slight. The arms were somewhat rigid. While the pupils were being examined again they were seen to dilate rapidly under the light, and in two or three seconds he had a tonic spasm, in which the head and eyes deviated to the left. The mouth opened and the left side of it was retracted. At the same time the left arm and leg were raised from the bed and partially flexed. This was succeeded by a slow movement of extension of these limbs. The tonic spasm then became general, and subsequently the clonic stage supervened. The pupils continued to dilate and the eyes to deviate to the left, even after the clonic movements had begun. The neck remained rigid after all movements had ceased, and the head was directed to the left. Within twenty minutes two similar fits occurred. Some hours later he had five more fits of much less intensity. The pupils were dilated, and there was prolonged tonic spasm of the limbs, but no clonic stage. In these attacks the head deviated to the right; the eyes and mouth were not affected. The commencement of one of these

seizures was marked by two or three movements of flexion of the right knee. This series of minor fits was followed shortly by an attack identical with that first described. Then ensued ten minor fits. On the morning of the next day (the convulsions having ceased for several hours) he was said to have been partially conscious, and nodded in answer to questions. The attacks soon recurred, consciousness being quite lost during the intervals. In the evening it was noted that he was lying on the left side groaning and passing his hand over the forehead as though in pain. There was no evidence of paralysis. He could not be roused to take notice. He died on the morning of April 15th, after being comatose for fifteen



A, B. Caudate and lenticular nuclei displaced and flattened out; with internal capsule between them.

hours. The temperature varied between 99·4° and 102·6°. To the foregoing clinical account I must add a few additional facts. The patient had never, so far as his friends were aware, been subjected to vomiting. He used to stammer, but I have ascertained that this dated from childhood. Dr. Sharkey informs me that he had from time to time examined his eyes and had not discovered any evidence of optic neuritis.

Post-mortem examination made by myself.—Body of a short, well-nourished man. Lividity marked on posterior surface, less evident anteriorly. Rigor mortis general and well marked.

The skull-cap was removed with some difficulty. There was an area of atrophy, two inches and a quarter in diameter, involving the right frontal and parietal bones just behind the

coronal suture, and reaching up to the middle line. The outer limit of this atrophied area corresponded to the coronal suture, and here there was a round perforation one-eighth of an inch in diameter. Immediately around this hole the bone was reduced to a mere shell. There was a corresponding degree of atrophy extending in an horizontal direction from the middle line of the frontal bone to the anterior inferior angle of the parietal bone. The frontal bone between these depressions was thickened and unnaturally vascular.

On the outer surface of the right side of the skull-cap there were two bosses, one corresponding to the anterior inferior angle of the parietal bone, the other to the atrophied area and perforation already mentioned.

The surface of the brain was much injected, but there was no lymph or stickiness anywhere. Occupying the white matter of the right hemisphere was a large, firm tumour. About its centre the growth came to the surface, and the dura mater was firmly adherent over it for a diameter of two and a half inches. The tumour bulged considerably to the left and had caused a deep depression on the median aspect of the left frontal lobe.

On making transverse vertical sections the growth was found to have the following dimensions :—

Pre-frontal	Section	2¼	inches	vertically	. . .	3¼	inches	horizontally.
Pediculo-Frontal	„	2½	„	„	. . .	3	„	„
Frontal	„	1¾	„	„	. . .	1¾	„	„

The growth extended nearly to the level of the tail of the caudate nucleus. The caudate and lenticular nuclei on the right side were displaced and occupied a horizontal position, as shown in the figure. On microscopical examination the tumour was found to be a sarcoma, composed chiefly of spindle cells. The organs of the body were normal.

Remarks.—This case requires little comment. It is possible that the head injury might have had some share in the causation of the morbid growth. Traumatism in my experience is far from uncommon as an antecedent in cerebral tumour, and I am inclined from a survey of several cases to ascribe some importance to injury as a causation. But apart from this, there is good reason for inferring that the growth in this patient was existent in 1877, when he had his first fit. This being so, the tumour must have had a duration of nearly ten years, death occurring in 1887.

The atrophy of the calvarium, as seen post-mortem, also points to the prolonged existence of the mass. It is certainly very remarkable that no symptoms of paralysis, or indeed of

any obvious intellectual disorder, should have been evident in this patient. The only definite indication of the brain mischief, looking back on the case, is the occurrence of convulsions, and little information, even had the existence of coarse disease been suspected during life, could have been drawn from them with respect to localisation. It is worthy of notice also that the symptoms resembled very closely ordinary epilepsy, in their occasional occurrence, in the supervention of mental disorder after the convulsions, and also in the amenability of the attacks to treatment by bromide of potassium.

I have since been thankful for the credit of cerebral surgery that the bosses on the skull had not attracted attention during life. It is clear from the condition of things seen at the autopsy that an attempt at removal would have resulted in failure, and possibly in death.

Critical Digest.

BRAIN AND EYE.—THE PUPIL.

BY JAMES ANDERSON.

The iris has been described specially by Iwanoff as possessing a sphincter and a dilator muscle, the former supplied by the third nerve or common motoroculi, the latter through the cervical sympathetic. This mechanism along with the contraction and dilatation of the vessels of the iris was held to explain the various pupillary states. Of recent years however there has arisen not only scepticism, but actual disbelief in the existence of a dilator muscle, Grünhagen in particular having strongly denied its existence. The question as to the nature of the radiating fibres in the posterior limiting membrane of the iris is admitted by microscopists to be one of much difficulty, but the preponderance of opinion seems to be distinctly against their being muscular. With the doubtful exception of the rabbit, the otter, and birds, a dilator pupillæ must be excluded, and it is clear if this be so that the dilator nerves of the pupil must supply the sphincter and be inhibitory in function. This is the conclusion reached by Gaskell in his recent work on the "Structure, Distribution and Function of Visceral Nerves"[1] where he says he "cannot see the urgent necessity for a special muscular dilator pupillæ; variations in the extent of the contraction or relaxation of the sphincter are quite sufficient to account for all the differences in the size of the pupil, if only the radial fibres of the iris possess, as Grünhagen thinks they do, a sufficient amount of elasticity." He quotes Grünhagen's experiments, showing that the suspended sphincter can be made to elongate by direct stimulation of the muscle itself, and draws a parallel between the iris and the intestine, as to muscular action and nervous supply. In the early part of the above-mentioned work he shows that the circular muscle of the intestine has a double nerve supply, viscero-motor through the accessory and vagus nerves, and a viscero-inhibitory through the abdominal splanchnics. The iris he holds is another example of a muscular structure supplied by two nerves of opposite character, the one motor, the other inhibitory; the motor by the common oculo motor, the inhibitory by the sympathetic trunk passing upward in the neck, which he terms "cervical splanchnic," and he finds that

[1] 'Journal of Physiology,' vol. vii., No. 1, p. 38.

the dilator fibres of it arise in the dog from the anterior root of the second thoracic nerve, an observation agreeing with those of Ferrier and Yeo in the monkey.

It will readily be seen that the above conclusions imply a most important change in our views as to the physiology of the iris, while at the same time observations on the pathology of the iris can be readily translated into the more modern terminology. The radical nature of the change is still more manifest when Gaskell's theory as to the real nature of inhibition is understood, that it is an *anabolic* action, diminished activity with repair of function; while the action of motor nerves is *Katabolic*, increased activity with exhaustion of function.

Fuchs has given[1] a careful account of the superficial anatomy of the iris, noting many new and interesting facts regarding it, which have a direct bearing on the above question. The appearances recorded by him can many of them be observed in one's own iris, as Priestley Smith points out by using lateral focal illumination and a concave mirror of very short focus to look into. The hand lens and oblique mirror of an ordinary refraction ophthalmoscope answer admirably. It will be seen that the iris is divided by a small circle into two zones, pupillary and ciliary. The former is crossed by elevated ridges converging toward the pupil and overlapped at its margin by the dark brown edge of the pigment layer. The latter is divided into three zones, the flat, the plicate, and the peripheral, the middle being marked by a series of nearly concentric furrows. The posterior limiting membrane, which must of course be studied in the dead eye, consists of fine radiating fibres showing no nuclei, and certainly, Fuchs believes, not muscular. When the pupil contracts, all the zones on the anterior surface of the iris, including the black border of the pupil, become broader. The sphincter widens, and the slit-like crypts in the neighbourhood of the small circle become longer and narrower. When the pupil dilates, on the other hand, the pigment border narrows or disappears, the crescentic furrows on the anterior surface deepen, and the crypts widen. The posterior limiting membrane is the only part of the iris not thrown into folds when the pupil dilates, and it is therefore presumably the only structure which takes an active share in producing this dilatation. The pigment layer being closely attached to this membrane is drawn back from the margin of the pupil. Were the dilatation caused by the contraction of any tissue lying in front of the posterior limiting membrane, the pigment layer would rather tend to protrude than to disappear.

In his Hunterian Lectures on the Intraocular Muscles of Mammals and Birds,[2] Jessop gives a good summary of the work recently done in this subject with illustrative and control experiments. He adopts Gaskell's view as to the structure and nerve supply of the iris, the short ciliary or myotic nerves derived

[1] 'Ophth. Rev.,' Jan. 1886. p. 11.
[2] Ibid. vol. vi., pp. 125, 159, 315.

from the third nerve, through the lenticular ganglion, the long ciliary or mydriatic nerves from the second dorsal nerve, as Ferrier showed in the monkey, and Gaskell has shown in the dog. These long ciliary or mydriatic fibres leave the central nervous system, as Gaskell shows, among the fine medullated fibres of the anterior root of the second dorsal nerve, pass by the cervical sympathetic, through the superior thoracic and the inferior cervical ganglion to the superior cervical ganglion. So far the fibres retain their medullary sheath, but here they lose it just as the abdominal splanchnics lose theirs in the semi-lunar ganglion, with which, and not with the chain of lateral or proximal ganglia, Gaskell considers the superior cervical ganglion to be serially homologous. From the superior cervical ganglion the mydriatic fibres seem to pass through the petrous portion of the temporal bone with the internal carotid artery to the anterior portion of the Gasserian ganglion, thus gaining the nasal nerve, from which they ultimately branch. Jessop notes that the mydriasis on ablation of the superior cervical ganglion is greater than on mere section of the cervical sympathetic below it.

The action of drugs on the pupil gives us most valuable information as to the physiology of the pupil, more especially the action of atropine, eserine, and cocaine. Jessop in his third lecture (*loc. cit.*) gives an excellent summary of these results. Atropine paralyses the unstriped muscular fibre of the eye-ball, eserine stimulates it, while cocaine stimulates the endings of nerves that relax the muscles. The combined action of any two of these agrees with these results. It is to be noted that atropine increases the mydriasis produced by section of the third nerve, ablation of the ciliary ganglion or section of the short ciliary nerves.

In a monograph of 79 pp.[1] Heddæus gives an account of the reaction of the pupil to light, its observation, measurement, and clinical signification. He asserts, and we believe with reason, that the observations and records of the state of the pupil, especially on the part of neurologists are extremely imperfect, and this he attributes partly to want of uniformity in the mode of observation, partly to the clumsiness and ambiguity of our nomenclature. In this last relation he recommends the uniform use of the terms "direct" and "consensual," the first to express the movement of the iris caused by variation in the amount of light falling on the retina of the eye in question, the second to express the movement produced in the opposite eye by that variation. The term "immobility of pupil" he would apply only to the pupil which has neither the direct nor consensual reaction to light nor the accommodative reaction. Excluding for the moment the reflex centre, it may be said that this condition of immobility implies disease of the centrifugal pupillary fibres. When the accommodative reaction is absent, the light reaction will almost invariably be lost. For the reaction of the pupil to light he suggests the terms "reflex sensibility" and "reflex insensibility" (Reflex-

[1] Abstract, 'Ophth. Rev.,' June 1887, p. 164.

empfindlichkeit, Reflextaubheit). Reflex insensibility of the retina, or what is the same thing if the media are proved clear, reflex insensibility of the eye, implies disease of the centripetal pupillary fibres.

The methods recommended by Heddæus for testing the pupillary reflex do not differ essentially from those employed by most careful ophthalmic surgeons, but as there is probably some ground for his reproach in the case of neurologists, we may enumerate them. In the simplest method, the patient stands about four feet from a window, looking out into the distance. The observer covers the left eye with his right hand for five to ten seconds, meanwhile watching the right eye. He now uncovers the left eye, and estimates the consensual reaction of the right eye. Again covering and uncovering the left eye, he observes the direct reaction of the left eye. He repeats these actions for the right eye. To exclude possible accommodative reaction, he must see that there is no convergence, and also whether the pause or latent period of the reflex, about 0·5 second, takes place. The pupil contracts to its minimum size with a jerk, widens slightly, and ultimately settles to its final size. If the reaction by this method be doubtful, Heddæus next partly covers the right eye until he can just see the outline of the pupil. He at the same time completely covers the left eye, and keeps both eyes thus covered for about a minute. Now uncovering the left eye, he watches the consensual reaction of the right eye and the direct reaction of the left. If the reaction is still doubtful, he sends the patient to the dark room for a quarter of an hour, the second eye is completely covered or bandaged, and the first is then suddenly exposed to the full daylight. For testing the reaction of different parts of the retina, as in hemianopsia, Heddæus employs the dark room and lamplight reflected into the eye with the ophthalmoscope. He has not satisfied himself of any difference in the reaction of the two halves of the retina in such cases.

The last part of Heddæus' monograph is occupied with an enquiry as to the course of the centripetal pupillary fibres, and he gives reasons for believing that all the fibres of the optic nerve do not serve for vision, but are some of them concerned in reflex action. Stilling and Gudden have asserted that this is so, and Gudden even states that the two groups of fibres are microscopically distinct, the visual fibres being smaller than the pupillary, and becoming atrophied on destruction of an anterior corpus quadrigeminum, while both the pupillary fibres and the pupillary width and reaction are unaltered. That the pupillary reaction to light may persist in bilateral total blindness has been known for some time, and von Græfe's explanation is generally accepted, that in such cases the cause of the loss of vision lies centrally from the point of divergence of the pupillary fibres. But sometimes the ophthalmoscope shows optic atrophy, and yet reflex sensibility persists. Here comes in the difficulty of diagnosing the degree of atrophy. In such cases it may be supposed that the nerves, although much damaged, are yet able to convey the light stimulus,

but that behind the point where the pupillary fibres leave the damage is so great as to destroy vision.

Heddæus considers from clinical evidence that the centripetal pupillary fibres run in the optic nerve with the macular fibres, and are more resistant to pathological processes than the visual. Also he agrees with Bechterew that these fibres probably do not pass into the optic tracts and do not decussate in the chiasma, but pass back behind the chiasma directly into the grey matter surrounding the third ventricle, and so reach the oculo motor nucleus of their own side. From this it would follow that dilatation of the third ventricle would cause dilatation of both pupils, and that unilateral destruction of the grey matter in the wall of the third ventricle would cause reflex insensibility on the same side. In support of his conclusion, that reflex sensibility is confined to the macular region, he points out that in deeply pigmented normal-eyed people illuminations of the peripheral parts of the retina may produce absolutely no pupillary reaction. And again, in unilateral mature cataract, direct illumination with the ophthalmoscope of the macular region shows reflex sensibility as higher in the sound eye, while lateral illumination shows it as higher in the cataractous eye. The result in the latter case he believes to be due to the diffusion caused by the lens opacity. If Heddæus' assertion be correct, it follows that in hemianopia there would be no difference in the pupillary reaction of the blind and the functional halves of the retinæ, and this, in opposition to Wernicke, he asserts to be the case. The observation of these cases is admittedly difficult and liable to many fallacies, but the weight of evidence is on the whole against Heddæus.

Steinach[1] has made some interesting observations on the pupils of vertebrate animals. He divides them into three groups, those having a total decussation of the optic fibres (horse, crocodile, &c.), those having a few non-decussating fibres, supplying, according to Gudden, peripheral parts of the retina (rabbit), and lastly, those having a considerable bundle of non-decussating fibres (man, cat, &c.). The first two groups show only direct pupillary reaction, even although they have partially overlapping visual fields, such as the owl. The third group shows both direct and consensual pupillary reaction. From the results in the first two groups Steinach implies first that the pupillary fibres of the oculo-motor nerves decussate, and second, that there is no intra-cerebral commissure between the centres of these fibres. It is clear from a comparison of the views of Heddæus and Steinach that farther anatomical and physiological observation is required before a conclusion can be reached as to the course of the pupillary fibres.

The observations of Hensen and Voelcker and of Kahler and Pick make it practically certain that the most anterior cells of the oculo-motor nucleus supply the ciliary muscle and the next posterior the sphincter iridis, and these cells are connected with the anterior fibres of the oculo-motor nerves. Westphal has recorded a case of

[1] 'Centralbl. für Physiologie,' 28 May, 1887, p. 105.

ophthalmoplegia externa with careful anatomical examination of the oculo-motor nucleus,[1] which practically settles this question, and accurately localizes in man the cells supplying the intrinsic muscles of the eye-ball. He shows that they form a median and lateral group lying dorsally from the general oculo-motor nucleus between it and the posterior commissure.

In 'BRAIN,' vol. i., Mr. Jonathan Hutchinson gives three papers on the symptom-significance of different states of the pupil, and since then many important papers have appeared in this journal on the subject. Uhthoff has recently published an important paper on the diagnostic value of absence of pupillary reflex.[2] His results, combined with those of Moeli, Thomsen and Siemerling, which he quotes, show that general paralysis and tabes dorsalis account for something like 83 per cent. of the cases of reflex pupillary immobility, showing the extreme value of this symptom in the diagnosis of these affections. It is present in somewhat more than half the cases of general paralysis, and Erb finds the light reaction absent or slight in something like 85 per cent. of the cases of tabes. Syphilis comes next in frequency as a cause of the loss of pupillary reflex, and in that case it is frequently combined with loss of accommodation, the ophthalmoplegia interna of Hutchinson.

Jonathan Hutchinson, jun., has recently made observations[3] on the pupil symptoms met with after injuries to the head, and he summarises his results as follows:—(1) In most cases of concussion, for a variable time, depending on the severity of the injury, the state of the pupil resembles that met with in ordinary anæsthesia produced by ether, chloroform, &c., *i.e.*, slowness to respond to light without marked myosis or mydriasis. (2) In a small proportion of cases of concussion temporary mydriasis (uni- or bi-lateral) is met with. (3) When inflammatory reaction follows severe bruising of the brain, myosis is the rule. (4) In compression of the brain, from meningeal hæmorrhage, mydriasis on the side of the lesion is met with in at least half the cases, double mydriasis occurring next in order of frequency, myosis being very rare.

Systematic and careful observation of the state of the pupils will undoubtedly in many cases assist materially in the diagnosis not only of nervous affections but of general diseases. An inequality or irregularity of the pupils, abnormal largeness or abnormal smallness of the pupils, should invariably lead to a careful investigation as to the cause. An old iritis may lead to the diagnosis of gout, rheumatism, or syphilis, which would otherwise have escaped us. The investigation of small pupils will save us from a diagnosis of rheumatism where tabes dorsalis is the real disease. Dilated insensitive pupils if recognised may save us from diagnosing glaucoma as dyspepsia, a not-unheard-of mistake, and one which few medical men would forget to their dying day.

[1] 'Ophth. Rev.,' 1887, p. 210. [2] Ibid. 1886, p. 104.
[3] Ibid. vol. vi., pp. 97, 129, 153.

Reviews and Notices of Books.

The Nervous System and the Mind. A treatise on the Dynamics of the Human Organism.—By CHARLES MERCIER, M.D., Lond. 1888.

THE author of this important work does not require a lengthened introduction to the readers of 'BRAIN,' the pages of which already owe to him many of its ablest contributions. It is well known that Dr. Mercier started upon his professional career as a pupil—we may, indeed, say, a favourite pupil—of Dr. Hughlings-Jackson; and in dedicating this work to his old master, he gives appropriate expression to the admiration he still feels for his teaching and character. The next most potent factor in the moulding of Dr. Mercier's mind is to be found in the philosophical writings of Mr. Herbert Spencer, which he has made the subject of prolonged and profound study. Any reader, then, who is acquainted with the writings of these two eminent men, will be well prepared to enter upon the study of the work before us; but, at the same time, we ungrudgingly acknowledge that Dr. Mercier has pondered upon psychological problems sufficiently long and arduously to have attained to the character of an original and independent thinker in his own right.

The work is divided into three parts, comprising the study of (1) nervous processes and physiological actions; (2) the psychological actions which eventuate in conduct; and (3) mind.

In the *first* part of the work the author deals, in the two opening chapters, with such physical problems as the nervous discharge and the nervous resistance. After showing the dependence of the nervous discharge upon the molecular complexity of the grey matter, with its *latent* energy ready to become *active* on the application of external forces, he enumerates the inductive laws of the discharge, giving, at the same time, a deductive interpretation of them in terms of molecular energy. The strength of the discharge, other things being equal, is directly proportional to the strength of the disturbing force or stimulus. Once began, it tends to become stronger and to spread; and its augmentation may be manifested either by an extension of the area of its action, or by an increase in its intensity. What now becomes of it? It finds a vent partly along accustomed and permeable channels, and is then expended chiefly in setting up muscular action, and partly along unaccustomed and more or less impermeable channels, and is then expended in overcoming

the resistance, and thus in developing new structure. The author believes that powerful currents escape from the nerve fibres into the ground substance; and if this be true, the molecular disturbance of this substance, which is most probably a form of connective tissue, is the correlative of the highest forms of consciousness. We do not believe that there is any justification whatever for this opinion, and think it more likely that the discharge is expended in increasing the permeability of the fibres which are already partially open, in lengthening the fibres which terminate in blind extremities, so that they come by-and-bye to form new connections with the fibres of adjoining cells, and in the formation of quite new cells and fibres. The author now shows that although the accumulation of energy in the grey matter is a more or less continuous process, and the resistance is a constant factor, yet when the nervous energy is being actively expended, it discharges itself in intermittent shocks; and he makes excellent use of this hypothesis in the explanation of the phenomena of tetanus, tremor, and clonic spasm.

In the physiological part of the first division, Dr. Mercier discusses the co-ordination of muscular contractions which constitute *movements*, and of the series of simultaneous and successive movements which constitute *definite acts*. *Co-ordination* is defined as combination in determinate ratio, and a *movement* as any set of co-ordinated muscular actions. Thus the term movement is made to include the muscular actions which maintain the fixed attitudes of the body as well as those which produce active changes of parts of the body in space. Viewed in regard to the region of the body concerned, movements may be divided into *central* and *peripheral*. The central movements occur in the trunk, and are continuous or tonic, indefinite, few in number, simple in character, and give rise to a coarse or general adjustment of the body; while the peripheral occur in the digits, mouth, and eyes, and are intermittent or clonic, definite, multiple in number, diversified in character, and give rise to adjustments which are very precise and specially adapted to particular ends. Passing now to the consideration of the higher co-ordination, by means of which a group of movements is arranged into a definite act, Dr. Mercier points out that the individual movements are either *simultaneous*, chiefly effected by the central muscles; or *successive*, chiefly effected by the peripheral muscles. The successive movements are of two kinds—*radial* and *lateral*. In the radial, the contractions begin in the central and spread to the peripheral muscles, or begin in the peripheral and spread to the central, these being respectively named *centrifugal* and *centripetal* radial movements. In the lateral, the succession is effected by the peripheral muscles, and the movement spreads from one physiological series to another, as from the arm to the leg, or from one digit to another. After some very suggestive remarks on inhibition, which the exhaustive discussion of the author's views in the last number of 'BRAIN' relieves us from criticising, and on the constitution of nervous centres in general, this part of the

work is brought to a close by the enunciation of Mr. Herbert Spencer's theory that the cerebellum is the great centre for the co-ordination of simultaneous, and the cerebrum for the co-ordination of successive movements; the former being the organ of compound co-ordination in space, and the latter the organ of compound co-ordination in time.

In the *second* division of the work, the author enters upon the study of the actions of the body, not as hitherto in relation to one another and to the body itself, but as they effect an adjustment of the organism as a whole to the whole of its surroundings or to its environment. This adjustment is effected neither by the co-ordination of muscular contractions which constitute a movement, nor by that highest co-ordination of movements which constitute a definite act, but by a highest co-ordination of definite acts to a common end, which constitutes *conduct*. It is by the observation of conduct alone that we can judge of the degree of intelligence of others; and in this study, which forms the subject-matter of *objective* psychology, no cognisance is taken of consciousness. The degree of intelligence is estimated by the novelty, the elaborateness, and the precision of the adjustments, and still more by observing whether the adjustments are or are not adapted to self-conservation, the rearing of offspring, and the welfare of the community. Excellent remarks follow on the nervous mechanism which underlies conduct and renders it possible.

In the third and probably most important division of the work, Dr. Mercier deals primarily with states of consciousness, these forming the subject-matter of *subjective* psychology. States of consciousness may be viewed wholly from an introspective standpoint, reference being made solely to their comparison with one another; or they may be viewed in connection with the circumstances in the environment with which they correspond. From the introspective standpoint, states of consciousness may be resolved into feelings, and the relation between feelings. So long as consciousness is occupied with a sound, for example, a simple feeling is experienced; but when the sound changes by becoming suddenly louder or shriller, the feelings of difference and sequence constituting the relations between the two sounds occupy consciousness, a judgment is formed, and the feeling has given place to a cognition or thought. But the primary division of states of consciousness into feelings and thoughts may be cut at right angles by a second introspective division of them into *vivid* and *faint* states. Vivid states are caused by objects present to the senses, and they are consequently named *presentative* feelings and thoughts; while faint states are revivals in consciousness of vivid states previously experienced, and are consequently called *representative* feelings and cognitions. In accordance with these divisions, Mr. Herbert Spencer classified states of consciousness into feelings and cognitions, while each of them are subdivided into serial order according to the descending degree of vividness with which they occupy consciousness, the most vivid being termed presentative, and the next in order, presentative-representative,

representative, re-representative, &c. The advantages Mr. Spencer claimed for this classification are that it represents, in a rude way, the order in which the feelings and cognitions are evolved, the presentative being common to all sentient beings, the presentative-representative to most animals and the human infant, the representative to the higher animals and the child, and the re-representative to adult man, while the feelings and cognitions which are still more remote from actual experience are peculiar to the most highly developed of the human race. It is at this point that Dr. Mercier diverges from his master. Without wholly rejecting the introspective classification, he prefers to regard states of consciousness with reference to the circumstances in the environment with which they correspond. From this point of view, a cognition or thought is the process of establishing relations in the organism in correspondence with relations in the environment. In accordance with this view, thoughts are divided into those in which there is (1) the establishment of a new relation, and (2) the revival of a previously established relation. When a new relation is established, the process is termed *reasoning*, and the result a *judgment* or *concept*. When a previously established relation is revived, if both terms of the relation are wholly represented, the process is termed *remembrance*, and the results are either *memories* or *concepts*; and if one or both of the terms of this relation contain presented elements, the process is termed *perception*, and the results, *percepts*.

Turning now to the feelings, they are divided from the introspective standpoint with reference to their development according to their degree of representativeness, quantitatively into intense or voluminous, and qualitatively into pleasures and pains. But without rejecting this classification, Dr. Mercier regards them as states in the organism which correspond with interactions between the organism and the environment. In accordance with this view, he classifies the feelings—

1. According to the way in which the interaction is begun, whether by the environment, by the organism, or by both.

2. According to the position of the respective feelings in the evolutionary scale, the order being from the low kind of feelings and emotions which affect the conservation of the organism, to the progressively higher emotions which affect the perpetuation of the race, the common welfare and the welfare of others, and to the still higher feelings which are neither conservative nor destructive, and those which correspond with relations between interactions.

3. According to the directness or indirectness of the correspondence of the feeling with the interaction, and the degree of remoteness from direct correspondence, the direct feelings being called *sensations*; the indirect, *emotions*; and the remote, *sentiments*. Following out these principles, the author arranges the feelings into six classes, which, with their sub-classes and genera, require for their representation no less than seventeen elaborately constructed tables. Dr. Mercier's analysis, with its accompanying

classification of thought and of the feelings, constitutes by far the most important as it is the most original portion of the work; and it seems to me now, as it did on reading it a few years ago in the pages of 'Mind,' to be incomparably the ablest contribution to subjective psychology since the publication of Mr. Herbert Spencer's great work.

This book forms such a solid and valuable contribution to psychology that we do not feel much in the humour for fault-finding. We will, however, venture to direct the author's attention to a few points in which the work is susceptible of improvement when the occasion arises, as we trust it soon will, for issuing a second edition. In the first place, we think that the introduction ought to be entirely re-written. Neither would the author's reputation lose anything, nor would his arguments be less forcible and convincing, did he write in a more courteous manner of his brother alienists, did he employ less bombastic epithets in his denunciation of the older psychological terminology, and, above all, were he to abstain from garnishing his sentences by the use of Biblical language. Dr. Mercier ought, in our opinion, to be much more sparing than is his wont of arguments of the sledge-hammer order. A sledge-hammer is a very effective weapon for cracking an opponent's skull, provided only his head be first fixed in the line of descent; but if it is not so fixed, the opponent steps aside, and laughingly observes the blow descend harmlessly by his side. As an example of this kind of argument we may quote Dr. Mercier's attempt to show that the proposition "that a causal relation exists between the events of mind and the movements of matter" is *nonsense*, in the literal meaning that it has *no sense*. Did the present writer maintain the truth of this proposition, Dr. Mercier would have no difficulty in cracking his skull, because he would himself make willing admissions respecting the theory of causation which would fix his head in the line of descent. But suppose that Dr. Mercier has to deal with an opponent who, following Hume, Stuart Mill, and Professor Huxley, regards causation as merely implying the existence of a definite order amongst phenomena established by experience, and who denies that there is in any case a community of nature between cause and effect, or a *nexus* which binds the one to the other, what would be the result of his sledge-hammer blow? None whatever. With such an opponent the author would soon find himself involved in a discussion respecting the theory of causation; and although he might still obtain a victory, he would feel himself under the necessity of laying aside his sledge-hammer and taking to the rapier. In other words, the only effective weapons in discussions of this kind are those which require for their use subtility and skill, and they cannot be safely replaced by exhibitions of mere brute strength. It is not maintained that Dr. Mercier is destitute of skill when he has the patience to exercise it; what is asserted is that he is lacking in sympathy with opponents. If they are not full-blown evolutionists, he does not even care to conceal his contempt for them, and he is much too fond of making a show

of his strength by attempting to overbear all resistance by one mighty blow.

The next point we notice is the halting interpretation the author gives of the terms "generality" and "speciality" as applied to movements (p. 94). Is any one the wiser for being told that "by a special movement is meant one which serves a special purpose, and that only"? Taken along with its context, some meaning might be read into this proposition; but even then it is far from being definite enough. This is to be regretted, because these terms are much abused in medical writings generally, where they are hardly ever used, as they ought to be, in the sense attaching to them in embryology, and in the classifications by species of botany and zoology. Generality means, according to Mr. Herbert Spencer, "manifestation in numerous cases;" and conversely speciality means manifestation in few cases. The movements of the trunk are met with in all vertebrate animals, and they are consequently very general. The peripheral movements are, avoiding details, peculiar to the higher vertebrates, and are therefore special; some of them are met with in man alone, and consequently they are still more special; while a few of them occur only in the most highly endowed and educated men, and these attain to the highest degree of speciality. The fact that Dr. Mercier has missed the true meaning of the correlative couple—general and special—has prevented him from making such fruitful use as he might have done of the comparative physiology and embryology of these movements. In his discussion of conduct, Mr. Herbert Spencer's classification of human activities might have been usefully introduced. He arranges them into—(1) "those activities which directly minister to self-preservation; (2) those activities which, by securing the necessaries of life, indirectly minister to self-preservation; (3) those activities which have for their end the rearing and discipline of offspring; (4) those activities which are involved in the maintenance of proper social and political relations; and (5) those miscellaneous activities which fill up the leisure part of life, devoted to the gratification of the tastes and feelings" ('Education,' p. 8). This classification, which is a most important one from the standpoint of alienism, would form the correlate, on the side of objective psychology, to Dr. Mercier's main division in the classification of the feelings on the side of subjective psychology.

The last point we shall notice is that, in his criticism of Mr. Herbert Spencer's classification of the feelings, Dr. Mercier is hardly fair. An author has a right to expect that his work should be judged according to the aim he has set himself to accomplish, and not according to an altogether different standard set up for him by his critic. Suppose an engineer, for example, erects a bridge for the purpose of carrying troops across a river, what should we think of a brother engineer who should criticise his construction as if it were intended as a permanent roadway for generations of peaceable citizens? Now the particular purpose for which Mr. Herbert Spencer advanced his classification of the

cognations and feelings was to arrange them in their due positions in the evolutionary scale; and the advantages he claims for his classification, which he distinctly states to be a provisional one, are that it is reached without a lengthened inquiry, and that it is "in harmony" with the order disclosed by detailed analysis. But that is not all. Mr. Spencer not only *must* know that in adopting a *serial* instead of a *specific* classification, he is using a very imperfect method, but he also distinctly states against his own classification almost every objection urged by his critic. The objections we have to urge against Dr. Mercier's strictures are that he criticises Mr. Spencer's classification as if it were intended to be a classification by species instead of a serial one; as if it were advanced as the result of a detailed analysis, instead of for its readiness of application; as if it were a completed classification, from which all objection had been removed, instead of being avowedly incomplete, and having most of the objections to it distinctly stated by its author; and as if it were regarded as a permanent classification, to be used for all purposes whatsoever, instead of being a provisional classification used only for a particular purpose. That Mr. Herbert Spencer had contemplated a very different classification of the feelings from this one, may be gathered from his own words. "We may," he says, "study the evolution of the emotions up through the various grades of the animal kingdom, observing which of them are earliest, and exist with the lowest organisation and intelligence; in what order the others accompany higher endowments, and how they are severally related to the conditions of life. In the second place, we may note the emotional differences between the lower and the higher human races—may regard as earlier and simpler those feelings which are common to both, and as later and more compound those which are characteristic of the more civilised. In the third place, we may observe the order in which the emotions unfold during the progress from infancy to maturity. And lastly, comparing these three kinds of emotional development, displayed in the ascending grades of the animal kingdom, in the advance of the civilised races, and in individual history, we may see in what respects they harmonise, and what are the implied general truths" ('Essays,' vol. i., p. 310). This passage requires no comment; and elaborate and valuable as we believe Dr. Mercier's classification of the feelings and emotions to be, it seems to us yet to fall considerably short of the ideal set forth in the above quotation. The great objection we have, indeed, to his classification is that the genetic relations of the higher and more compound to the lower and simpler emotions find only an imperfect expression in it; but we shall look for this defect to be removed in the further development of the author's system. It would be an injustice to Dr. Mercier not to mention that all he claims for his classification is that it is "founded on the principle of evolution, and that it harmonises with Mr. Spencer's psychology, of which it is, indeed, an extensive corollary." This statement is sufficiently modest; but we still think that a

good deal of the criticism he directs to Mr. Spencer's classification is misdirected, and might have been spared.

But whatever may be the faults and shortcomings of this book, it cannot be denied that it is a noble contribution to psychology. Its intrinsic merits are great, and it is still greater in promise. For it must be remembered that the present volume only forms an introduction to the author's complete scheme, and that upon it as a foundation he intends to erect a system of scientific alienism.

Several of the distinctive questions of alienism are discussed in this volume with sufficient fulness to enable us to form a tolerably accurate opinion of Dr. Mercier's views with regard to them. He will doubtless object (p. 213) to the distinction usually made between emotional and intellectual insanity. He, however, admits that in normal consciousness the emotions and thoughts, especially in their higher forms, are inextricably blinded. Yet this does not prevent him from separating thought and feeling in his classification of mental states. In other words, he allows the practical utility of this distinction to override all theoretical considerations; and when he comes to deal more specifically with the morbid phenomena of consciousness, he may find the retention of the old division of insanity into moral and intellectual practically advantageous, whatever may be the theoretical objections to it. The author's opinion of the particular disorder of the mind which constitutes insanity, may be gathered from his analysis of thought and his subsequent remarks. When once we pass beyond the purely introspective view of consciousness, thought is the establishment of a mental relation in correspondence with a relation in the environment. In insanity, the mental relations, when considered amongst themselves, may be valid; but their correspondence with external relations is not only unadjusted, but is incapable of being adjusted (p. 250). Here we come within sight of a scientific definition of the terms delusion, illusion and hallucination, which will replace the imperfect and often absurd meanings given to them in current writings. The writer is tempted to reproduce here definitions of these terms which he gave in a course of lectures delivered in the early part of last year. Dividing knowledge into that which is obtained directly through the senses, and that which is *indirectly* established by reasoning, it may be said that hallucinations and illusions are a disorder of the former, and delusions of the latter. *Hallucinations* and *illusions* are *unreal* perceptions, the presented factors in the former being generated in the organism, and of the latter in external circumstances. In insanity there is in addition a conviction of the reality of these perceptions which cannot be corrected by experience. A *delusion* is primarily a belief in a proposition, the truth of which, if challenged, could only be established by a process of reasoning. In the next place the proposition does not correspond to fact, and it is consequently a *false* belief. If, however, strong reasons exist which would give semblance of truth to the proposition, we should hardly call it a delusion, and consequently we must amend our definition by

saying that a delusion is a false and *groundless* belief. But before a delusion is reached, another essential constituent must be added, that the belief in the truth of the proposition is incapable of being corrected by experience, and we now reach the ultimate definition of a delusion as a false, groundless, and *incorrigible* belief. We cannot enter here upon a defence of these definitions; but if the terms used be translated, as is easily done, into Spencerian language, it will be found that they do not differ much from Dr. Mercier's views.

We have said that we did not feel much in the humour of fault-finding, and yet have gone on fault-finding until we have become quite censorious. The author will, we trust, believe us when we say that the reason and justification for our fault-finding is to be found in the very excellence of his work. In the volume before us is laid in broad lines the foundations of a system of alienism which, when completed, will, judging from the author's comprehensive grasp of his subject, challenge the admiration of future generations, and will be read and studied when the fleeting publications with which the medical press is teeming are placed on the shelf and forgotten. Dr. Mercier's work is, in short, far too good for a critic to content himself with a simple analysis of its contents, followed by a few platitudes and indiscriminate eulogy. We believe that amongst the younger generation of the profession are to be found a great many earnest, thoughtful, and learned men, to whom this volume will be most welcome, and who will carefully read, re-read, and ponder the detailed psychological analysis, the comprehensive generalisations, and the valuable hypotheses which are so lucidly expressed in its pages.

JAMES ROSS, M.D.

Physiologie des Exercices du Corps. Par le Dr. FERNAND LAGRANGE. 1 vol. pp. 372. Paris. Alcan, 1888.

THIS volume of the "International Scientific Series" is calculated to direct very timely attention to a subject of great importance. Calisthenics are just now the fashion, and a book dealing methodically with the rationale of muscular therapeutics was much wanted, in order that the limits of this mode of treatment may be appreciated by the profession and the public. Dr. Lagrange's work forms part of the "International Scientific Series," and we may confidently anticipate the appearance of an English edition at no distant date. Although the work is presumably written for intelligent readers of all classes, and takes the form of a popular exposition of the subject, we have no hesitation in strongly recommending its perusal to our readers. Dr. Lagrange has an admirable grasp of his subject, and writes so tersely and lucidly, and has so many new ways of presenting old facts, that few can read his work without instruction and none without pleasure.

Dr. Lagrange begins with an excellent account of the physiology of muscle, nerve, spinal cord, and brain in relation to movement. The facts he lays before the reader in these sections are indispensable for obtaining a philosophic grasp of the subject, but need not detain us as they are all certainly familiar to the readers of 'BRAIN.' The author does well, however, to dwell upon the fact that the least movement performed by the human machine sets many of its wheels in motion. "To move the forearm, the arm needs to be fixed, the arm must be steadied on the shoulder, the shoulder on the thorax and spinal column; but these being supported by the pelvis and legs, it follows that the whole body must co-operate to move the forearm." This is a fact too often forgotten, but it needs to be constantly borne in mind when considering the localisation of the so-called cerebral motor centres. Again, it is well to insist that "In performing a movement a muscle never acts without the simultaneous contraction of its antagonising muscle, which serves, as it were, as a counterpoise and check."

"Effort" is defined as that arrest of respiration which takes place during vigorous muscular acts for the purpose of giving support to those muscles which are attached to the thorax. "Effort," however, is often seen in acts where the expenditure of force is on the whole small. It is seen whenever it becomes necessary to contract a group of muscles with all the force of which they are capable. Look at a man cracking walnuts with his fingers. If the fingers be strong and the shell brittle, the muscular contraction is limited to the forearm, the face remains calm and the respiration is undisturbed; the nut is cracked without effort. But if the man has to use all his power to break the shell, the muscular contraction spreads from the forearm to the arm, shoulder, neck, chest, and abdomen; breathing stops, the face reddens, the veins of the neck and face swell, and when the shell at length gives ways a kind of groan is heard, like that of the porter who lifts a burden from the ground. Some surprise may be felt that an act involving directly only the flexors of the fingers should necessitate the contraction of such distant muscles, and that in order to squeeze with the hand the abdominal muscles become essential. But the body being composed of many movable pieces, and no muscle being able to put forth all its strength unless one of its ends be rigidly fixed, it follows that for the fixation of the forearm the arm, shoulder, thorax, and pelvis must be steadied. This end cannot be attained unless a great mass of air is in the lung, for the lung distended with air is the only support for the movable bones of the thorax. The distended lung forms a sort of resisting cushion upon which the ribs rest firmly as they are pulled downwards by the abdominal muscles. The extra flow of blood to the contracting muscle is the main factor which produces the increased activity of the circulation which is one of the initial phenomena of muscular exercise, and which produces an increased activity of vital processes in every organ of the body inclusive of the brain. The Peripatetics disputed as they walked about, and J. J. Rousseau has said, "Movement and

walking favour the play of the intellect and facilitate the work of thinking." Exercise, indeed, may produce excitement not unlike drunkenness, as is seen in the dancing dervishes, or without going so far, may cause a great flow of animal spirits, as is seen every day in the ball-room.

The fatigue of a muscle, as seen in the laboratory, when contraction no longer follows stimulation, is caused by the paralysing effect of the chemical products of contraction. This form of fatigue probably never occurs in nature except in the case of hunted animals, and when thus caused is sufficient of itself to produce death. The painful sensation of fatigue is due to impressions made upon the intramuscular nerves, and occurs long before the physiological exhaustion of the muscle. It is a sensation which preserves us from the deadly effects which would result from the presence in the blood of an excessive quantity of the chemical products of muscular work. The sensation of fatigue is a cerebral phenomenon, and its occurrence is largely influenced by the amount of cerebral involved by any particular act.

One of the commonest results of active exercise is breathlessness, or as athletes say, getting "blown." This condition is one of considerable interest, and Lagrange deals with it at length under the name of *essoufflement*.

The intensity of breathlessness is in direct proportion to the amount of force expended in a given time. It is a sort of physiological measure of the amount of work. When the amount of work done is very great, as when a man runs up four pairs of stairs, and thus raises 140 lb. through 50 feet in a minute (or let us say 7000 foot-pounds, or more than 3 foot-tons, in a minute), breathlessness is necessarily produced with great quickness. The exercise of a great mass of muscles loads the blood with carbonic acid, and when the production of carbonic acid is greater than the eliminating power of the lung, the respiration gets troubled and the worker is "blown."

Exercises of the arms, with a muscular mass much less than that of the legs, are slow to produce breathlessness, and the arm-worker is apt to get tired before he is "blown." When hard work is done with the legs, however, the reverse holds good, as the runner is often "blown" before he is tired.

If, during exercise, an "effort" as defined above be made, and the respiration be temporarily arrested for the purpose, it follows as a necessary consequence that "loss of wind" is the sooner produced.

Again, physical or emotional irritation producing reflex effects upon respiration, such as the sudden impact of cold, fear, anger, or laughter, by checking the eliminative power of the lung tend to hasten loss of wind. The man who scolds or whips a willing horse so as to produce fear during work effectually defeats his object. There is a striking resemblance between a man in a state of terror and one "blown" by exercise.

In the early stages of exercise, pulse and respiration are alike quickened, and there is a determination of blood to the lung. As

a result of this the elimination of carbonic acid is increased, and the effects of exercise are compensated. If the respiration, however, be unduly quickened, the true function of the lung is lessened and carbonic acid accumulates in the blood. The hurrying ventricles of the heart also fail to compensate by rapidity what they lose in force, and thus is brought about a condition of things physiologically similar to asphyxia—passive congestion of the lung, a distended right heart, and a left ventricle tending to increase in feebleness. When the excessive exercise inducing this alarming state ceases, Lagrange has observed that the respirations reach the normal sooner than the pulse, and the following is the mean result of observations made upon himself and two guides during an ascent of Mont Canigou :—

	Pulse.	Respiration.
Complete Repose	62	14
During the most severe climbing	123	30
Five minutes after reaching summit . . .	117	14

According to Lagrange excessive exercise causing breathlessness alters the rhythm of respiration. During repose the times occupied by inspiration and expiration are, as he says, exactly equal, but observations made on a man who has run himself out of breath show that the rhythm has completely altered, inspiration being much longer than expiration. His explanation of this fact is as follows: The first effect of exercise is the quickening of the blood current, and the active congestion of the lungs. The need is felt of disembarrassing the lung, and to this end the aspirating effect of inspiration is brought to play. This gives comfort to the breathless man; but when the blood current slackens during expiration the lung is again engorged, and hence the quick instinctive return to inspiration. The lung has need of disencumbering itself of blood and carbonic acid; the one is favoured by inspiration and the other by expiration, and thus the "blown" athlete is placed upon the horns of a dilemma.

A person completely "blown" is in a critical condition, and many of the deaths which take place on the race-course are due to a fatal syncope supervening on the asphyxia caused by loss of wind. If we are to get good results from exercise, and free from all danger of overstraining the heart and lungs, we must choose such moderate exercises as do not involve an excessive amount of work or the making of "efforts." Loss of wind it need not be said depends, as to the time of its occurrence, very largely upon training and the state of health.

There are other effects of exercise which are not quite so immediate as breathlessness. After unwonted exertion a man is apt to feel stiff and "knocked up," and this condition is described by Lagrange under the name of *courbature*. It is characterised by sleeplessness and restlessness, notwithstanding a feeling of being tired out, and this is accompanied by some heaviness in the head, a coated tongue, loss of appetite, and stiff and painful muscles. Lagrange asserts that exceptionally this condition is

accompanied by fever, but he does not give any details as to temperatures, &c. The liability to be knocked up varies with the state of health, and reaches the vanishing point with a man "in training." One of the most common symptoms of this condition is the formation in the urine of a deposit of urates, and the circumstances which favour these deposits have been carefully studied by Lagrange on himself and others.

When a healthy man takes some exceptional exercise the appearance of his urine varies with the time of its secretion, the amount of exercise and the state of training. Lagrange asserts that the effect of exercise on the urine is not immediate, and that it is useless to expect the appearance of urinary sediments earlier than three hours after exercise. Other things being equal, it is asserted that the urinary sediments are directly proportional to the work done by the muscles. "According as the exercise is more or less violent the precipitate may vary, from a slight cloud appearing only once to a thick muddy deposit lasting twenty-four hours." The liability to the precipitation of urates lessens with training until no amount of work seems capable of producing it. But, and this is noteworthy, if a given exercise for which the athlete is in training be discontinued and another be substituted involving the use of different muscles, the liability to *malaise*, stiffness and urinary deposit reappears. "The man accustomed to hard walking feels no trouble after a long tramp, but he will be knocked up by a short bout of fencing if he be unaccustomed to that exercise. An examination of the urine will show that this fluid, although it remain clear after a twelve-hours' walk, becomes cloudy with urates after twenty minutes with the foils." It is almost needless to say that any impairment of health increases the liability to *courbature* and the accompanying urinary sediments. Lagrange claims to have proved that the urinary deposits are not due to a concentration of the urine as a result of heavy sweating. He gives an interesting account of how he rowed with a friend (both being at the time in perfect training) from Limoges to Paimbœuf, near the mouth of the Loire, a distance of 550 kilomètres, with the additional labour of dragging the boat over the locks, eighty-three in number. The work done was very excessive, and the food very substantial, with lots of wine and coffee. Yet they were never knocked up, and the urine, examined every day, never showed any sediment. If these sediments be the result of sweating, surely they should have occurred on Aug. 10, the second day of their journey, when they rowed 50 kilomètres under a broiling sun and slept at night beneath a feather quilt in a room over a baker's oven. A slight mishap to the boat gave them leisure on the following day for repeated urinary examinations, but "in spite of sweating, which had soaked our garments by day and our bedclothes at night, no urinary deposit occurred in my friend or myself."

An analysis made by Mr. Papon of the urine of a man out of training after a long bout of fencing, gave 1·43 grams of uric acid per litre, while the urine of the same person after training,

and after the same amount of exercise, gave only 0·60 grams per litre. The *malaise* of fatigue is largely due, according to Lagrange, to a species of uræmia, and the well-known fact, that unwonted exercise often determines an attack of gout, seems to give support to this theory. The fact that training renders the liability to this condition very much less is probably due to the fact that the man out of training has his muscles encumbered with tissues of reserve, which disappear as his training advances.

Just as breathlessness is due to a self-poisoning by carbonic acid, so knocking up from fatigue is due to a self-poisoning by waste products from the muscles. As for the stiffness and tenderness of the muscles, Lagrange is inclined to attribute these to slight physical injuries of the fibres of the muscles and tendons.

A man or animal overworked becomes jaded, and this ultimate result of fatigue is discussed under the name of *surmenage*. An animal hunted to death may die from asphyxia and syncope as we have seen, but the hunted animal which escapes is not unfrequently found dead in its lair on the following day, and this death is due to an auto-intoxication by the waste products of muscular work.

Excessive muscular work produces well-recognised changes in the muscle. The sudden onset and rapid disappearance of rigor mortis in hunted animals is well known. The gourmet appreciates the tenderness and flavour of the coursed hare, and the cook knows that a coursed hare is "fit" much sooner than one shot, and that it is very liable to go putrid. Jaded muscles yield on analysis an undue proportion of extractives, and also, it is said, of alcaloidal bodies. It is very rare, except perhaps occasionally on the field of battle, that men die from acute muscular overwork, but a jaded condition, the result of "all work and no play," is not uncommon. It is asserted that the process of self-poisoning by waste products may cause a kind of typhoid fever, and Lagrange is careful to assert that it is something more than a mere predisposition to the fever, but an actual "auto-typhisation" (to use an expression of Professor Peter's), which is brought about. This requires more investigation. The disease which follows upon "forced marches," and the liability of overworked and jaded troops to succumb to various forms of ill-health, is, however, perfectly well known. Lagrange asserts that the *miasme humaine*, or androsmia, is far more marked after excessive work, and he alludes to the overpowering smell of a barrack-room which has served as a dormitory for troops after a forced march.

It is asserted also that most cases of "sunstroke" are really largely due to muscular overwork. When excessive exercise is accompanied by mal-nutrition we get a condition of wasting and chronic exhaustion, in consequence of the tissues of reserve being used for physiological combustion.

Nervous exhaustion is one of the results of muscular overwork, as it is of intellectual overwork. Lagrange states that he has seen many cases of transient melancholia occurring at the beginning of autumn, and due to the excessive labour of the harvest. As a

rule the rustic is not called upon to make any great physical exertion, but from the beginning of June he often labours inordinately, working very long hours, and putting forth all his strength; his sleep is short, his diet is often unsatisfactory, and the penalty which he pays is often that of nervous exhaustion.

The ideas of J. J. Rousseau that fresh air and pure morals are a sure protection against diseases of all kinds are not tenable.

Peasant women are very liable to exhaustion, for, in addition to great physical labour, they have to endure their maternal causes of exhaustion. Nervous women are as common in the country as the town, but the manifestations of their condition are less obtrusive. They have no time for crises, but neuralgias, gastralgias, vertigoes and neuroses of every kind form the stock complaints of the female peasant exhausted by physical labour.

Fatigue results from (1) certain material changes in the organs of locomotion (slight bruising, &c.); (2) self-poisoning by waste products; (3) absorption of living tissues tending to produce wasting; and (4) dynamic exhaustion of the motor elements of nerve and muscle.

Rest has for its object the removal of the effects of fatigue, viz. (1) the cessation of the discomfort caused by the material changes in the organs of locomotion; (2) to give time for the getting rid of the waste; (3) time for repair; and (4) time for the nerve and muscle elements to gather energy.

Aptitude for exercise is one of those things that grows by what it is fed on, and, provided time be allowed for repose and the subject be well nourished, the more work a man does the easier he will do it. It is a common expression that the man in training is as "hard as nails," and this hardness of the tissues is no figure of speech, but a sober fact. As the hands get protected by a growth of epithelium, and soon cease to blister and bruise as the result of manual labour, so the deeper tissues, muscles, bones, synovial sacs, ligaments and nerve twigs soon cease to suffer from the friction of exercise.

By exercise we become disencumbered of superfluous fat, the heart and lungs perform their functions with greater power and regularity, and the nutrition of the body is improved throughout. The effectiveness and ease with which muscular acts are performed increases with practice, and, although it is not capable of demonstration, it is certain that an improvement in the nutrition of the nervous system must take place. It is certain that physical courage is to a large extent bred of physical training, and that thus one mental quality follows to some extent in the wake of bodily work.

Exercises may be divided for purposes of classification into violent, moderate and gentle; and, with regard to these, Lagrange offers a timely word of warning. The quantity of exercise taken is often difficult to measure. When a man walks upstairs, it is comparatively easy to calculate the work done in foot-tons or pounds; but, when a man is struggling with an adversary, the calculation is by no means so easy. For the same individual the

best index of work done is the physiological effect produced on the lungs and heart. A violent exercise produces breathlessness before fatigue, a moderate exercise will produce local fatigue before breathlessness, and gentle exercise will scarcely produce either the one or the other. We are warned against the error of measuring the amount of work done by the difficulty of the exercise. Thus, a man who walks ten feet upstairs, and another who raises himself to the same height by a rope "hand over hand," do the same amount of work; but the muscular mass involved in the act being in the latter case relatively small, the difficulty is great, and local fatigue is liable to stop the performance. In the easy amusement of skipping, a little girl really takes a very large amount of exercise, whereas in a gymnasium she gets often a very inferior amount of exercise, and is bothered rather than amused in trying to learn some tricky *tour de force* which, when accomplished, does not produce a physiological effect in any way equal to the skipping-rope. At the present time it is the fashion to regard the trapeze as the regenerator of the human race, and we are told by gymnastic pedants that it is only by long research and profound meditation that we can use our limbs. Even in young ladies' boarding-schools it is the fashion to erect gymnastic machinery and teach difficult and grotesque exercises, notwithstanding that many a childish game produces a far greater physiological result.

Lagrange arranges his exercises in three classes :—

(1.) *Exercices de Force*, which might be called massive exercise, such as carrying a heavy load upstairs or wrestling. These exercises quickly induce fatigue and loss of breath, because the local effect on the strongly contracted muscle is severe, and the carbonic acid of which the system seeks to be rid is necessarily enormous.

These exercises demand a great expenditure of muscle, but they favour repair. They involve very little nervous excitement and very little cerebration, such as is involved in feats of skill.

The muscular contractions are powerful, slow, and sustained, and these, according to Lagrange, are very favourable for the nutrition of the muscles, because the highly oxygenated blood is long in contact with them. These exercises stimulate largely the organs of respiration and circulation, and, as they do not tax the mind or emotions, they are favourable to nutrition, and those who practise them are often herculean in form. On the other hand, in subjects who are out of training or weakly they lead to a variety of muscular mishaps and encourage emphysema and lesions of the vascular system.

(2.) *Exercices de Vitesse.*—Rapid exercises often involve an amount of work quite equal to the massive exercises, and like them they encourage the full expansion of the lungs and quicken the circulation. In a bout of fencing or a spurt of racing, the work accumulated soon equals that of the miller's man who toils up a ladder with a sack of meal, or the wrestler the whole of whose strength is antagonised, and who, in spite of supreme and continued effort, produces very little visible effect. All of these

take their exercise in "big doses." Painful fatigue is not so marked in rapid as in massive exercise, because it is evident that four movements of ten kilogrammes each do not strain the muscles as does one movement of forty kilogrammes.

Rapid exercises do not make the muscles grow to the same extent as massive exercises, because, it is said, the afflux of blood to them is not so great. They cause a great development of the chest, however, and the big thorax and small calves of the professional runner are well known. In rapid exercises the response of the muscle to stimulation becomes phenomenally quick. This is partly due to the training of the muscle, but partly also to the force of the stimulation, or, in other words, to the degree of mental effort. When we "hurry," the mental effort is at its maximum.

The fatigue brought about by rapid exercise is different to that due to massive exercise, and the feeling of it is akin to that caused by moral excitement. It does not conduce to repose or sleep, and in fact seems at times to oppose them, and hence it is that nutrition after rapid exercise is less marked than after massive exercise, and those who pass their lives in hurrying work are generally thin and have smaller muscles than those who labour hard and slowly, notwithstanding that the sum of work accomplished may be the same in both cases. Competition and hurry cause great nervous excitement, and this often has the effect of putting a man or an animal off his feed. It is the racehorse or hunter which refuses its corn at night, and not the drayhorse.

Lagrange devotes a chapter to (3) *Exercices de Fond*, by which he means those gentle exercises which are practised habitually and more or less continuously. As a type of this class of exercise one may take canoeing or walking on the level. These exercises do not cause fatigue nor breathlessness, because the elimination of carbonic acid keeps pace with its formation. They do not produce a great development of the chest, because the lung is not so fully distended as in rapid exercise. These exercises are to be recommended for persons with weak lungs or heart.

Although there is no accumulation of carbonic acid, the same cannot be said of the nitrogenous waste from the muscles, which tends to accumulate, and that to a large extent, if the exercise is long continued. After a long day's shooting the man out of training feels more or less "done up" on the following day, notwithstanding that his exercise may never have produced breathlessness. It is alleged that the nitrogenous tissues in the young are very unstable, and tend unduly to accumulate during prolonged exercise, which is very trying indeed to children, whose natural aptitudes fit them for rapid exercise.

In analysing the various exercises in vogue, Lagrange points out that the tendency of the age is, in gymnastics, to put the arms to their wrong use. Trapezes, parallel bars, and rigging are the stock apparatus of the gymnasium. On these the whole weight of the body is moved by the muscles of the upper limbs, and the relatively small muscular mass and the unstable humero-scapular

joint effect with difficulty what the massive legs and solid hips and pelvis do easily.

These exercises do not create the same need of breathing as do those in which the big mass of the lower limbs is involved; they are difficult and their execution is worrying, and they produce an excessive development of the muscles of the shoulders and nucha, which is in fact a deformity. It is a mistake to suppose that these exercises produce great chest development: to do this a large mass of muscle must be set in motion so as to cause the breathing to be deep. It is the runner who gets a big thorax, and not the mountebank of the trapeze, who to a moderately developed chest adds a mass of shoulder muscle which makes him look almost humpbacked.

One of the peculiarities of trapeze and bar exercises is the frequent necessity of flexing the pelvis on the trunk, a movement which is very rare in any of the habitual exercises of life. This is done mainly by the recti abdominis, which become highly developed in professional gymnasts. Trapeze and bar exercises, by increasing the firmness of the abdominal wall, prevent the growth of the belly, and, according to Lagrange, such exercises are the very best *ceinture contre l'obésité*.

The general effect of exercise is to stimulate nutrition and increase metabolism. Elimination of waste products is the first result, but in a healthy individual this waste is more than counterbalanced by quickened absorption. It therefore results that plethora is reduced while the appetite is increased. Hence we generally see, as the effect of well-regulated exercise, that the fat persons regain their figures, and the thin and ill-nourished put on flesh. The really healthy person feels a need of exercise, which should take its place along with hunger and thirst as one of the natural appetites of the body. This appetite, if not obeyed, languishes, and the lazy person becomes so encumbered with surplus tissues that movement becomes painful, and the taking of necessary exercise gets more and more difficult in proportion to our abstinence from exercise. Laziness, as we all know, is a dangerous vice which is apt to grow upon one, and the effort to throw it off becomes more and more difficult with its continuance. Lagrange combats the notion, which is very general, that the best way to "develop the chest" is to exercise those muscles part of whose function it is to elevate the ribs. It is no doubt true that well-trained and thoroughly exercised chest muscles are an advantage, and the human figure does not assume its ideal form unless the muscles of the thorax and upper limbs are well developed; but Lagrange is undoubtedly correct when he maintains that the degree of inflation of the lungs is the main factor in determining the capacity of the thorax, and, if exercise is of a kind to produce deep inspirations of the lungs, the thorax will grow big no matter what muscles are mainly in use. Inspirations are deepest when the mass of muscle in use is biggest, and, as the muscular mass of the lower limbs is greater than that of the upper, it follows that running and similar exercises are likely

to cause, and in fact do cause, great development of the thorax, *i.e.* increase in size of the thoracic cage. Gymnastic machinery for the development of the chest is a mistake, and if a little patient has a narrow, retracted chest, it is best to recommend running or a skipping-rope.

The abuse of gymnastic machinery—trapeze, parallel bars, &c.—which all throw the support of the body upon the upper limbs, tends to produce an ungraceful figure and genuine deformity. The shoulder muscles are developed enormously, the upper dorsal region of the back is arched and the scapulæ tilted and dragged forward. The professional gymnast gets to look hump-backed and top-heavy. Fencing, if carried to excess, especially by young subjects, is liable to cause a slight lateral curvature of the spine towards the sword arm, and to counteract this it is advisable to use the left hand occasionally.

French boxing, it is alleged, is a far better exercise than fencing, *i.e.* for a young man. French boxing involves kicking, and thus all four limbs are abundantly exercised as well as the spinal column, which is ever moving to maintain the balance of the body, when the combatant attempts the *coup de pied* with either foot, and especially when he first of all kicks his adversary in the face with his right foot, and then delivers a second blow on the same spot with his left foot by means of the *coup de pied tournant*. Undoubtedly this French edition of the "noble art" would make the practiser as sharp and lissome as a monkey.

Among exercises which may safely be recommended, is rowing, especially with a pair of sculls, because the development of the body is perfectly symmetrical.

For the improvement of the figure and for the encouragement of an erect bearing, all exercises requiring an accurate balance of the body are recommended. It is also essential that both sides of the body should be brought equally into play. The carrying of light weights upon the head is recommended as one of the best modes of improving the figure.

It would appear that in France as in England the question of "Overpressure in Schools" has occupied the public mind, and the Academy of Medicine has passed resolutions to the effect that the scholars in elementary schools are in need of less sedentary work and more physical development. Lagrange does well to point out that there is a strong analogy between mental fatigue and muscular fatigue, and that the brain is, so to say, the organ of exercise as well as of thought, and that it is by no means always true that during physical exercise the mind is at rest.

The mental and nervous effort is often out of proportion to the visible effect. The cat prepared to spring, the fencer on the alert, watching his adversary with a sudden movement ready planned in his mind, are by no means at rest, and it is well known that fencing is an exercise which causes great nervous exhaustion, which of course is enormously increased when the foils are naked and the "play" is in earnest. In men whose nervous energies are taxed by study, fencing is not a desirable exercise, but it is ex-

cellent for those who spend a life of idleness, and who need some exercise which shall stir their minds as well as their muscles. It is, says Lagrange, an excellent exercise for those who wish to get thin or to keep thin, as the constant nervous tension helps to this end. It is noticeable that the cat has less tendency to get fat than other domestic animals, and he attributes this to the fact that it is ever on the watch, prepared to spring on any object, be it mouse, rat, fly, or what not.

Some exercises require far greater delicacy of co-ordination than others, and the perfectioning of some acrobatic movements is more a matter of co-ordinative education than of muscular development. Many a *tour de trapeze* is easy enough "when you know the way," and, although it worries the learner, the physiological result is small from the point of view of muscular exercise. For those who are working hard with their muscles exercises of the simplest kind or such as have already been fully learnt should be chosen. It is very undesirable to harass children with a drill-master or gymnast during the hours which ought to be devoted to "play." Dancing, says Lagrange, is an amusement, but "learning to dance" taxes both mind and body. He draws a graphic picture of the terrible ordeal undergone by a distinguished oculist while learning the polka. The brows knit with his mind concentrated on his legs, the feet moving regardless of rhythm, and the sweat rolling off his forehead. "Never," said he, "was I equally bothered by an operation for cataract."

While some exercises bother the mind and demand (at least at first) a great amount of attention, others, such as walking, rowing, and many childish games, are performed almost automatically. These simple exercises are those which are best for the scholar working hard for examination. Such an one ought never to be pestered with gymnastic machinery and acrobatic tricks. For the indolent person, with his intellectual faculties lying fallow, exercises which demand attention, and have in them a certain element of difficulty, are to be recommended.

G. V. Poore, M.D.

Intracranial Tumours. By Byrom Bramwell, M.D. Edinburgh: Young J. Petland, 1888.

This book is difficult to criticise, for the author has either avoided introducing much debatable matter, or has alluded to all the views concerning the subject under discussion. The result is that he has presented us with a remarkably clear and concise exposition of the principal facts known about intracranial tumours. Each symptom is explained upon the basis of physiology, by which means that dry catalogue of symptoms so common in text-books on medicine is avoided, and any senior student after reading the book would feel that he had an accurate rational knowledge of the

subject. We are afraid, however, that the excellence of the book will prevent its having a very wide circulation, for cerebral tumours are not sufficiently common in general practice for the practitioners to care to obtain such an elaborate book as this is, consisting as it does of 270 pages and 116 figures, whilst the fact, that many of the more doubtful points are barely alluded to, deprives the book of the value it might have been to those who are specially interested in nervous diseases.

The author is to be congratulated on having all the way through the book clearly distinguished between those symptoms which point to the existence of a tumour, and those which indicated its exact site. This distinction is one which students frequently fail to grasp.

We are fully in accord with Dr. Bramwell in his belief that the general symptoms of cerebral tumour, such as headache, vomiting, double optic neuritis, vertigo, mental dulness, lethargy, &c., are due to the increased intracranial pressure caused either directly or indirectly by the tumour. As evidence of this pressure he adduces the separation of the cranial bones that one sees in young children in whom they are not yet firmly united. We are rather surprised that he has not mentioned the remarkable thinning of the bones of the skull seen in cases of cerebral tumour, even when the tumour does not come in direct contact with the bone. Cases have been recorded in which the thinning of the bones from this intracranial pressure was so great, that they could be pressed in like the bottom of an oil-can. The reasons why sometimes localising symptoms are fallacious are fully considered, but, as Dr. Bramwell says, the danger arising from these fallacies has been probably overestimated.

The Chapter on the causation of Double Optic Neuritis in Cerebral Tumours is a particularly clear and unbiassed account of the rival theories. The favoured view is that of Leber and Deutschmann, which is that the essential and primary cause is the presence of an irritant in the cerebro-spinal fluid, a secondary and favouring cause being the increased intracranial pressure.

The value of the work to those specially interested in nervous diseases would have been much increased if there had been a fuller discussion on the controversy about the usual and auditory centres, on aphasia, and on the rise of temperature sometimes seen in cases of cerebral tumour. The chapter on the differential diagnosis will be found extremely useful to the student of nervous diseases, for in it all the possible mistakes are considered, and by reading the chapter carefully he will learn to have them always before his mind. Attention is specially directed to the fact that tumours of the centrum ovale may produce symptoms like those of a cortical tumour, because they grow towards and irritate the cortex. This, although well known, is sometimes forgotten in cases of cerebral disease. In alluding to the greater frequency of tumours of the brain in males than in females, Dr. Bramwell adopts the usual suggestion that this may be due to the fact—the men following more dangerous occupations than women, are more liable to head injuries; but he makes no mention of a fact which must also be

borne in mind, namely, that tumours of the brain are commoner in male than in female children, and one may fairly assume that the two sexes are in children equally exposed to head injuries.

The book is, if anything, too well got up, the number of figures and their size adds to the bulk of it, and presumably to the cost of it, and therefore renders it less available to students. Many of the illustrations of the pathological nature of the tumours might have been left out, for such illustrations are common enough in pathology books, and some of those showing the position of tumours might have been much diminished in size.

We can most thoroughly recommend the book to those who wish to get an accurate knowledge of the subject; so clearly is it written, that we wish there had been more of it.

HALE WHITE, M.D.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology.—Titles and Indication of Contents of recent Papers. By ALEX. HILL, M.D., Master of Downing College, Cambridge.

ANATOMY.

1. Models illustrative of Brain Growth and Cranio-Cerebral Topography. CUNNINGHAM (*Lancet*, 1888, p. 1028. *Dublin Journal of Med. Sci.*, 1888, *No.* 8, p. 151.)

2. The Topography of the Brain. J. M. CHARCOT (*Forum, New York*, v. pp. 613–626, 1888).

3. Structure of Brain. LUYS (*L'Encéphale*, vi. p. 513, vii. p. 16, viii. p. 129).

General survey of present state of knowledge.

PHYSIOLOGY.

4. New Ideas with regard to the General Physiology of the Nerve Centres, explaining the Pathogeny of Paralysis and Anæsthesia. BROWN-SÉQUARD (*Comptes Rendus Soc. de Biol., Paris*, p. 277 and p. 290, 1888.)

PHYSIOLOGY—BIRD.

5. Physiology of Bird's Brain. SCHRADER (*Pflüger's Archiv*, xliv. *Nos.* 5 and 6, pp. 175–239, pl. iii., *Nov.* 1888.)

The effects which follow removal of great brain.

COMPARATIVE ANATOMY—ANNELIDA.

6. Central Nervous System of Lumbricus. FRIEDLÄNDER *Zeit. f. wiss. Zool.*, vol. xlvii. pt. 1, pp. 47–85, pl. ix. and x., *September* 1888).

COMPARATIVE PHYSIOLOGY—FISHES.

7. The Functions of Central Nervous Systems and their Phylogeny. Part II., Fishes. STEINER (*Brunswick, Vieweg and Son*, 1888; 27 woodcuts, 1 lithograph).

The volume already published on the brain of the Frog deals with function only. Present volume considers anatomy and phylogeny also. Various functions dealt with in ten chapters:

(1) With regard to equilibration, fins removed or thrown out of function; these organs do not preside over locomotion, which depends upon pendulum-like swinging of the tail. Fishes without paired fins are never seen swimming free in water. They serve to arrest quick movement and produce backward movement. Equilibration depends on general muscle-sense. (2) Bony fishes. Skull-case opened and closed again. Bones of skull lifted from in front. Replaced. Wound closed with gelatin, which is coagulated with strong solution of tannin. In two days gelatin-cap falls off wound, by this time united (Squalius cephalus). Fore-brain removed; normal movements; able to see obstacles; appearance of volition; worm seized and swallowed one day after operation. Removal of roof of mid-brain: blindness. Removal of base of mid-brain: fish falls on back; no locomotion except on mechanical stimulation. Hind-brain cut behind lobus impar: locomotion lost; respiration ceases. (3) Amphioxus may be divided into two or three parts, each of which balances itself and moves with head-end forwards: metameric locomotor centres. (4) Scyllium catulus and canicula: fore-brain removed; locomotion as in bony fishes, but inability to ingest food; death from starvation. Cerebellum removed: no disturbance of movement on equilibration. Section above vagus origin; no locomotion. Below vagus origin: trunk makes normally balanced movements, lasting for hours, or even a whole day. (5) (6) (7) (8) Spinal cord in roach, ganoids, petromyzon, and eel. (9) "Locomotion centre" in medulla. Forced movements depend upon activity of general locomotion centre. Forced movements after one-sided injury of those parts of the brain in immediate relation to locomotion. (10) In cartilaginous fishes same results follow removal of fore-brain and of olfactory lobe: conclusion that their fore-brain is nothing else than olfactory centre. Similar phylogenetic relation of fore-brain in all vertebrates. Brain comes into existence for purpose of associating at least one higher sense organ with locomotion centre.

Embryology.

8. Atlas d'Embryologie. Duval (*Paris, Masson*, 1889, 40 plates).

Embryology—Spinal Ganglia.

9. Morphology and Development of the Nervous System of Vertebrates. Beard (*Anatom. Anzeiger*, iii. pp. 874–898, *November* 7 and 17, 1888.)

A sketch of principal results obtained by author and published *in extenso* elsewhere. Maintains *contra* His that the "zwischen Strang" is a portion of the epiblast, which takes no share in the formation of the cranial or spinal ganglia, and that "zwischen Rinne" does not exist; on the contrary, there is in the head as in the trunk a "zwischen Strang." With regard to the essential point, the origin of the spinal and cranial ganglia for epiblastic

structures outside and independent of the central nervous system. Beard and His are at one, although differing as to exact mode of growth. This origin of the ganglia and dorsal roots from metameric rudiments situate at the extreme dorsal part of the epiblast, and re-entering angle between the dorsal portion and the neural tube, is true for all sensory nerves, including the olfactory. Each typical cranial ganglion arises from two sources, the "neural ganglion" (in the situation just described) and the "lateral ganglion," developed in connection with the branchial sense organs. In frogs the lateral ganglia separate from the epiblast before the neural ganglia reach them. The neural plate in all vertebrates, amphioxus being excluded until further investigated, is a paired structure, the two halves being separated in the median line by a ciliated groove, as in developing annelids.

EMBRYOLOGY.

10. Development of the Peripheral Nervous System of Vertebrates. (Part I. Elasmobranchii and Aves.) BEARD (*Quarterly Journal Microscopical Anatomy*, cxiv. pp. 153–228, pl. xvi.–xxi., *October* 1888).

Work *in extenso* of which the abstract is given in the *Anzeiger* quoted above. Previous accounts have all, with certain partial exceptions, described the posterior roots as growing out from the primitive spinal cord in the same way as anterior roots. Beard shows by numerous sections of embryo torpedo, pristiurus, mustelus, chick, &c., that the ganglia, spinal and cranial, originate independently of the primitive spinal tube, and that the posterior roots and sensory nerve fibres grow inwards towards the spinal cord, and outwards towards the periphery from the cells of the ganglia. Details of views and points of departure from His, Froriep, and Onodi, too complicated to abstract.

EMBRYOLOGY—NEURENTERIC CANAL.

11. Neurenteric Canal and Anal Canal in Rabbit. GIACOMINI (*Archives Ital. de Biologie*, x. 2, pp. 273–294, plate, *September* 1888).

MORPHOLOGY.

12. Development of Brain Mantle in the Animal Kingdom. EDINGER (*Deutsche Med. Wochenschr.*, 29, p. 604, 1888; *Münchener Med. Wochenschr.*, 27, p. 457, 1888).

GREAT BRAIN—CONVOLUTIONS.

13. Convolution of Broca in Primates. HERVÉ (*Bull. Soc. d'Anthropologie, Paris*, xi. p. 275, 1888, plates).

14. Convolutions of Great Brain. SCHNOPFHAGEN (*Wiener Med. Presse, No.* 45, pp. 1624–5, 1888).

15. Broca's Convolution. HERVÉ (*Paris*, 1888, *Lecroisnier et Babé*, pp. 165, 10 figs., 4 plates).

Cortex—Localisation.

16. Brain of Man from whom Limb had been amputated. Arnozan (*Journal de Méd. de Bordeaux*, xvii. p. 553, 1887–8).

17. Functions of the Occipital and Temporal Lobes of the Monkey's Brain. Brown and Schäfer (*Phil. Trans. Royal Soc., London*, vol. 179, B., pp. 303–327, pl. 48–50, *August* 1888).

Extensive ablations with knife and cautery; in some cases all the parts removed at once, in other cases after successive intervals. Favourable recovery in all cases. Animals kept alive for weeks or months, then killed, brain examined: lithographs of brains.

Unilateral removal of occipital lobe: immediate and persistent blindness for all images falling upon the corresponding half of each retina. First bilateral operation: complete blindness without any recognizable changes in eye-balls; second case, blindness persistent, but not complete; lower parts of retinæ being still sensitive (small portion of external surface of right lobe was left). The angular gyri were not injured in these cases. Angular gyri removed; sensibility of conjunctiva not affected.

Temporo-sphenoidal lobes on one or both sides, partially, or completely removed. In all cases, same negative results. No sensory disturbance. In two of these cases, a remarkable disturbance of intellectual functions. Animal became for a time idiotic. Movements slow. Senses dulled. Memory very defective. Former experience lost. Slow to acquire new experience. Things, animals, and persons deliberately examined with all the senses. Animals fed with head in dish instead of picking food up. Gradual but complete recovery.

Cortex—Secretion.

18. The Influence of the Cortex upon the Secretion of Saliva. Bechterew and Mislawsky (*Neurologisches Centralblatt*, 1888, 20, pp. 353–356, *October* 15).

Dogs curarized to prevent mistakes owing to contraction of facial muscles. Secretion from submaxillary gland, when anterior part of fourth convolution, bordering sylvian fissure (corresponding roughly with face centre), is stimulated with current too weak to affect other cortical centres. More extended region for stronger current.

Cortex—Function.

19. Function of the several Layers of Ganglion Cells in the Sigmoid Gyrus of the Cat. Von Monakow (*Arch. d. Sci. phys. et nat.*, xx. 10, pp. 358–360, *October* 1888).

Corpus Striatum.

20. Physiology of the Sub-Cortical Ganglia and their Relation to Epilepsy. Ziehen (*Deutsche Med. Wochenschr., No.* 27, p. 457, 1888, *and other journals*).

Great Brain—Fornix.

21. Olfactory Bundle of the Cornu Ammonis. Zuckerkandl (*Anat. Anzeiger*, iii. 15, pp. 425–434, *June* 1888).

Foville and others have observed fibres from anterior pillar of the fornix streaming towards olfactory lobe. Structure of this part of the fornix in osmatic animals (sheep, pig, &c.), compared with that of man and other primates. In the pig the "olfactory bundle of the cornu ammonis" courses in the body of the fornix to the back of the septum pellucidum: here, like the pillars of the fornix, it bends down, but passes in front of the anterior commissure [pre-commissural fibres of Huxley], on the mesial surface of the hemisphere, towards the base of the brain, where it "streams into the temporal and olfactory lobes."

Epiphysis.

22. Development of Pineal Gland. Francotte (*Archives de Biologie*, viii. 4, pp. 757–821, 1888).

Corpora Quadrigemina.

23. Optic Lobes of Teleostei and Corpora Quadrigemina of more highly organized Brains. Auerbach (*Morphol. Jahrbuch*, xiv. 3, pp. 373–394, pl. xvi. *Nov.* 13, 1888).

Crus Cerebri.

24. Fibre Systems and their Degenerations in the Pes Pedunculi Cerebri. Sioli (*Allgem. Zeitschr. f. Psychiatrie*, xl. 4, pp. 428–431, 1888).

Mid-Brain—Post. Long. Fasc.

25. Constitution of Posterior Longitudinal Bundles (Fasciculus Longitudinalis Posterior). Jakowenko (*Wjestnik Psich.* 1888 : *in Russian*).

A case in which these bundles as well as a great number of other parts of the brain were degenerated. Only where the adjacent nuclei and central grey matter were also diseased could the degeneration be traced. It ascended—uninterrupted from oculo-motor nucleus to posterior commissure. These latter fibres not considered to be continuation of those farther back. Conclusion that greater number of fibres are short commissural; the long fibres probably sensory.

Optic Chiasm.

26. Incomplete Crossing of Fibres in Optic Chiasm of Dog. Vitzou (*Comptes rendus, Acad. des Sciences*, cvii. 12, pp. 531–533, 1888).

Cerebellum.

27. Ontogenesis of Cerebellum. Lahousse (*Arch. de Biologie*, 1888, i. p. 43).

28. Degenerations consecutive to Extirpation, total or partial, of the Cerebellum. Marchi (*Rivist. sper. di Freniatria*, xiii. p. 446, 1888).

Completion of previous experiments on dogs.

Conclusions: removal of one-half of cerebellum produces degeneration of medulla of same side and olive of opposite side, and to slight extent of olive of same side, and medulla of opposite side. Superior peduncles do not cross completely. Middle peduncle not true commissure, but fibres lose themselves in grey matter of pons.

One-sided extirpation also induces degeneration of direct cerebellar to certain extent of pyramidal fibres of same side. Degenerated fibres are also found in cranial nerves and anterior roots of spinal nerves, and partial fatty degeneration of musculature of same side.

Cerebellum—Ataxy.

29. Cerebellar Locomotor Ataxy. Borgherini (*Riv. sper. di Fren.* xiii. 4, 1888).

30. Case of considerable Destruction of the Vermis Cerebelli, and Discussion of so-called Cerebellar Ataxy. Becker (*Virchow's Archiv*, cxiv. 1, pp. 173–186, pl. v., *October* 1888).

Monticulus, folium cacuminis, tuber valvulæ and pyramis of vermis replaced by two cystic tumours. Patient, eighteen years old, died of pneumonia. She never exhibited any evidence of vertigo, paralysis, disturbance of sensation, head-ache, or reeling gait.

Olive.

31. Minute Structure of Medullary Olive. Vincenzi (*Atti R. Acad. Med. di Roma*, 1888, pp. 171–182, 3 plates).

Medulla Oblongata.

32. Obscure but important Phenomena exhibited by a Dog after Section of one-half of the Medulla Oblongata. Brown-Séquard (*Comptes rendus Soc. Biol., Paris*, 1888, p. 407).

CORD—CENTRES.

33. Centrum ano-vesicale. ROSENTHAL (*Wiener Med. Presse*, 18, 19, 20, 1888).

Situation of this centre in man subject of considerable dispute. R. localises it in sacral cord, from observations and experiments on case in which bladder only was paralysed.

CORD.

34. Methods of investigating the Topography of the Motor Routes in Spinal Cord, &c. STEFFAHNY (*Beit. zur Anat. und Phys., Eckhard*, xii.).

SPINAL CORD—HISTOLOGY.

35. A Nucleus of Encapsuled Nerve-cells in the Antero-lateral Column of Human Lumbar Cord. CONTI (*Giorn. C. R. Accad. di Medicina, Turin*, 1888, *No.* 7).

REFLEX TIME.

36. Latent Period for Cerebral Irritation and Duration of Reflexes. NOVI and GRANDIS (*Boll. di sc. Med. di Bologna*, 1888, p. 5).

REFLEX—EYELID.

37. Centre for reflex Closure of Eyelid. NICKELL (*Pflüger's Archiv*, xlii. p. 547).

Hinder boundary of centre in the middle of ala cinerea, or perhaps somewhat higher. "Ascending" root of fifth, therefore, nothing to do with this reflex. Anterior boundary front of pons.

NERVES—CRANIAL—OPTIC.

38. The Central Ending of the Optic Nerve in Vertebrates (*Zeit. F. Wiss. Zool.* xlvii., pt. 1, pp. 1–47, pl. i.—viii., *September* 7, 1888).

Method: tissue placed in osmic acid $\frac{1}{2}\%$ to 1% for from 14 to 20 hours; sections cut in 70% alcohol; washed, placed in 80% alcohol 3 or 4 hours; mounted in water; ammonia run in under the cover glass. The section which is usually cut by hand very thick, becomes on the addition of the ammonia as clear as glass, the darkly-stained medullated nerves being thus rendered very easy to follow. Ammonia can also be applied to sections cut on a microtome in celloidin; they can then be mounted in balsam. Numerous illustrations of sections taken from Podarcis muralis, Lacerta viridis, Trapidonotus natrix, Emys Europæa, Testudo græca, Rana esculenta, Bufo vulgaris, Triton cristatus, Siredon pisciformis, Carassius auratus, Macropodus viride-auratus, Anguilla vulgaris, Lepadogaster Gouanii, sparrow, Parus major, hen,

dove; Mus musculus, Mus decumanus albus, Cavia cobaya, Erinaceus Europæus, Lepus cuniculus.

General results: fibres followed to their real ending in the "corpora opticum" (tectum opticum and corpora quadrigemina anteriora). In some types optic fibres pass through and cross over in the tuber, but return to optic tract. They are mixed with fibres of (1) commissura inferior; (2) fibræ ansulatæ; (3) decussatio inferior of birds (perhaps part of system 2); (4) numerous other thalamus fibres which make complicated plexus in mammals; (5) peduncular fibres which traverse the tract in neighbourhood of chiasma; (6) fibres traverse corpus geniculatum thalamicum, but do not end there; numerous other thalamic and cerebral fibres are mixed up with optic fibres in this region. Various strata of zonal fibres have to be distinguished. Object of paper to distinguish the primitive and proper optic fibre from those various tracts which take advantage of same routes.

II. Morphology of corpus opticum. Grows from upper wall of middle cerebral vesicle. Corpus posterius (quadr. post.) belongs rather to hind-brain. Originates in lateral intermediate region between mid- and hind-brain. The posterior tubercles of corpora quadrigemina of mammals are derivatives of corpus posterius of lower vertebrates.

Nerve—Cranial—Auditory.

39. Striæ Acusticæ in Man. Virchow (*Arch. f. Anat. u. Phys.* 1888, 3 and 4, p. 392).

Demonstration by means of transverse sections of medulla that in early life the stria acustica forms a strong bundle recognizable with the naked eye, running from the auditory nerve to the middle line, where it plunges into the raphe. It causes a local enlargement of the raphe, in which it seems to run far ventralwards.

40. Conductor Sonorus (*Bergmann*), and Origin of Acoustic Nerve. Nussbaum (*Med. Jahrbüch*, 1888, 5, p. 187–195).

41. Conductor Sonorus, and Remarks on Origin of Auditory Nerve. Nussbaum (*Med. Jahrbücher*, 1888; also *Neurol. Centralbl.* 1888, 24, p. 673, *December* 15).

The bundle which ascends from the striæ acusticæ received above name, with German synonym Klangstab, from Bergmann. By Stilling it was regarded as inconstant root of trigeminus. N. cannot decide between these two connections; describes bundle as surrounding in its course a round group of ganglion cells, originating in tegment, crossing to opposite side in dorsal part of raphe, and passing partly to floor of fourth ventricle, partly to locus cæruleus.

Nerves—Cranial—Vagus.

42. Central Ending of Vagus and Connection with Fasciculus Solitarius of Lenhossek. Bechterew (*Wjestnik Psichiatrii*, 1888, 2: *in Russian*).

43. Anatomy and Physiology of Vagus Nerve. Dees (*Archiv f. Psychiatrie*, xx. 1, pp. 89–102, pl. 1, *October*, 1888).

Literature of the several origins assigned to vagus.

Litter of new-born rabbits: vagus exposed in neck, cut and peripheral portion torn out. Animals killed at fifth week. Vagus to next branch above section normal thickness, but greyish red, and evidently principally connective tissue. Sections of medulla stained in aniline-black. Atrophy on same side of nerve-cell of (1) dorsal vago-glossopharyngeal nucleus, (2) ventral vago-glossopharyngeal nucleus. Shrinking of solitary fasciculus. Cells of ventro-medial portion of dorsal nucleus completely destroyed. Concluded from this, that axis-cylinder processes of these cells go to motor nerves of same side, for thorax; that N. intermedius Wrisbergi does not arise from proximal portion of the dorsal vago-glossopharyngeal nucleus. Ventral vago-glossopharyngeal nucleus lies between olive and lateral column. Well-defined group of cells; continuation caudalwards of facial nucleus, although separated by nerve tissue. Bundle goes directly backwards to solitary fasciculus. "Nervus accessorius vagi is not the motor laryngeal nerve." The ventral vago-glossopharyngeal nucleus is the nearest centre for the innervation of the muscles of the larynx. Fasciculus solitarius is the ascending sensory root of the N. vagus et glossopharyngeus. No evidence with regard to origin *viâ* the raphe. No origin of vagus fibres from funiculus teres.

44. Vagus Extirpation. Dees (*Neurologisches Centralblatt* 1888, 21, p. 596, *November* 1).

Note to paper by author in *Arch. f. Psychiatrie*, xx. 1.

Nerves—Peripheral.

45. Nerve Supply of Mouth and Nasal Fossæ in Vertebrates. Gaupp (*Morphol. Jahrbuch*, xiv. 3, pp. 436–490, pl. xix. *November* 1888).

Nerves—Peripheral—Sympathetic.

46. Sympathetic System of Fishes. Chevrel (*Comptes rendus, Acad. des Sciences*, cvii. 12, pp. 530–531).

47. Some Points in the Physiology of Gland Nerves. Bradford (*Journal of Physiology*, ix. 4, pp. 287–316, *November* 1888).

Histogeny.

48. Histology of Fœtal Brain. Magini (*Atti Accad. dei Lincei, rendiconti*, iv. 12, pp. 760–764, 1888, plates).

49. Genesis of specific-Nerve cells and the intimate Structure of the Central Nervous System of Birds. Falzacappi (*Bollett. Soc. di Naturalisti in Napoli*, ii. 2, pp. 185–193, 1888).

HISTOGENY—CORTEX.

50. Development of Elements of Cortical Layers of Cerebrum and Cerebellum. VIGNAL (*Arch. de Physiologie*, xx. 7, pp. 228–255, pl. v. and vi., *October* 1888).

Method of hardening; early fœtal brains; equal parts alcohol and osmic acid; also Flemming's and Henneguy's methods. Second half fœtal life: *alcool au tiers* (32° centésimaux) twenty-four hours; picro-carminate of ammonia (1 per cent.) two or three days; osmic acid, twelve to twenty-four hours. Literature. Brains of rabbits; brains of human fœtus, five and a half months; two sorts of nuclei colouring to different depths, but otherwise similar. Darker nuclei found of larger size; belong to pyramidal cells. Seven months: three layers; (1) 250 μ thick; (2) 1237 μ cells in series vertical to surface. At the junction of these two layers are found, the developing large pyramids with all the characters in miniature of the adult elements, homogeneous protoplasm staining deeply in osmic acid, irregular, giving off processes; large, round or oval, clear, dark-bordered, granular nucleolated nuclei. In matrix surrounding these cells, smaller nerve nuclei and neuroglia nuclei. These cells at the seventh month correspond to the large cells of the spinal cord at four months.

51. Ditto. *Continuation* (No. 8, pl. ix.–xii., *November* 15, 1888).

Adult neuroglia cells of cortex resemble neuroglia cells of the cord in the fifth month of intra-uterine life. They do not become distinct until end of sixth month. Seventh month easily freed from surrounding matrix; they possess a spherical or ellipsoidal dark-bordered nucleus, staining pale rose in picro-carmine. After this, neuroglia cells difficult to dissociate from matrix of cerebellum. In early periods, the superficial layer is full of closely packed nuclei, belonging, as the author believes, to migratory cells. Clear layer to be found between this layer and "granular layer," in which Purkinje's cells make their appearance at sixth month. The numerous hæmatoxylin-staining nuclei V. regards as belonging to neuroglia; rarer larger eosin-staining nuclei as nervous.

HISTOGENY—MEDULLA.

52. Development of Medulla Oblongata. HIS (*Archives des Sciences physiques et naturelles*, xx. 10, p. 356, *October* 1888).

HISTOLOGY—NEUROGLEIA.

53. Interstitial Tissues of Central Nervous System and Cerebro-spinal Nerves. PETRONE (*Gaz. degli Ospitale, Milan*, 1888, *Nos.* 4 and 11).

HISTOLOGY—NERVE ENDINGS—TENDON.

54. Termination of Nerves in Tendons in Vertebrates. PANISINI (*Riforma Medica, Rome*, iv. p. 752, 1888).

Trzebinski on the Action of various Hardening Methods upon Ganglion Cells. (*Arch. für. Path. Anat. u. Physiol. u. für. Klin. Med.* Bd. cvii. H. 1.)—The occurrence of feeble staining, vacuolation, clouding and granular disintegration in the ganglion cells of the spinal cord of dogs and rabbits that had been poisoned with phosphorus or arsenic was described by Popow and Danilo as being the direct result of that poisoning. More recently Kreyssig has observed exactly similar conditions in the spinal ganglion cells of healthy animals as well as in animals subjected to the lethal action of those substances.

Finding those specified changes were absent in specimens that were examined by the fresh method, Kreyssig was led to attribute them to the media used for hardening the tissue.

Professor Flesch has in great measure confirmed Kreyssig's observations. Nissl still later has declared that the ganglion cells are altered by Müller's fluid, but are unaffected by alcohol.

The author has investigated the question by systematic experiments, using the spinal cord of healthy dogs and rabbits, and of a rabbit which had been submitted to phosphorous poisoning for eight days.

The methods of hardening were:—

1. In Müller's fluid four or five weeks: the tissue was then either washed and transferred to absolute alcohol (or 10 per cent. alcohol for five days, then absolute), or it was put into alcohol, and kept dark, without previous washing.

2. In alcohol: absolute from the commencement, or weak at first and gradually increasing the strength.

3. In chromic acid: the first six hours in 0·1 per cent., then in 0·25 per cent., for forty-eight hours, subsequently in alcohol, or a mixture of alcohol and Müller's fluid.

4. In 10 per cent. mercuric perchloride solution for eight days, afterwards in alcohol containing 0·5 per cent. of iodine.

Borax carmine, alum carmine, Weigert's hæmatoxylin, and magenta red were the principal stains employed after the chemical treatment: the fresh preparations were coloured with methyl green.

All the last mentioned (dissociated elements and frozen tissue sections) gave results corresponding with those of Kreyssig: the ganglion cells on the whole showed no marked difference in the intensity of their staining; they had no pericellular spaces, and such details of structure as granules and striæ or fibrillation could be recognised. On the other hand, in all the hardened prepara-

tions, the ganglion cells manifested striking changes in the form of pericellular spaces, vacuolation, or other alterations of the cell substance, both with respect to its intimate structure and its receptivity for various colouring agents.

The pericellular spaces were largest and most numerous in the specimens which had been hardened in alcohol alone. They were larger and more irregular in the spinal cord of dogs than in that of rabbits. Sections from different parts of the same cord, after identical preparatory treatment, presented great diversity with regard to the number and size of the spaces: even in the same section the individual ganglion cells were unequally affected. Numerous and extensive pericellular spaces would be found in one anterior horn, while in the opposite corner they were sparse and ill-defined. Sometimes they were large in the cervical and lumbar regions, scarcely perceptible in the dorsal. They in general were less marked in connection with the pale tinted cells than around the deeply stained.

The bridging over of the pericellular space by extremely delicate filaments from the shrunken protoplasm, as described by Flesch, was often seen, more especially in Müller's fluid or chromic-acid preparations.

Vacuoles were observed, in small number, in specimens hardened either in chromic acid, Müller's fluid or alcohol—most abundant in the first named. None existed in the fresh tissue, nor in that hardened by corrosive sublimate. No vacuoles were discovered in the nuclei of any of the preparations.

1. In preparations hardened after Müller's method, or any modification thereof, the cell protoplasm was indifferentiated, homogeneous, and possessed, when stained with borax carmine, a peculiar vitreous lustre, calling to mind the appearance of hyaline or amyloid tissue.

After due allowance for obvious sources of error in estimating the degree of tinting, there remained a considerable number of ganglion cells which resisted, more or less completely, all the staining reagents used by the author.

The contour and minute structure of the nuclei came out clearly when not obscured by over-staining. The degree of their coloration bore no constant relation to that of the cell substance.

2. When hardened in alcohol, the cells were sharply contoured: granulation or striation of their substance could generally be recognised. In the latter case the striæ—deeper tinted than the

clear ground substance—usually coursed from the periphery towards the nucleus, leaving, however, a free zone around it.

The differentiation of the cell structure was clearly brought out by alum carmine or magenta red, and was analogous to that noted in the fresh tissue. Large pale ganglion cells were rarely found; the protoplasm of those existing appeared coarsely granular. The nuclei tended to be more distinct than those of the preceding category: they usually were less deeply stained than the body of the cell, and allowed one or more darkly tinted nucleoli to be perceived.

3. The nuclei of the ganglion cells in chromic-acid specimens were very clear in outline and minute features. On the other hand, the cell bodies themselves were obviously more changed in these preparations than in all others. This was shown by the large pericellular spaces, the coarse granular differentiation of the cell protoplasm, and the relatively frequent presence of marginal and genuine vacuoles. Partial or complete disintegration of some of the ganglion cells, indistinctness of their contour, and clouding of their substance, also were more commonly seen than in other preparations—some nuclei being surrounded by granular detritus, others lying free.

Pale cells were less numerous than in category 1.

4. The corrosive sublimate process gave results similar in many respects to alcohol preparations, but the shrinking of the cell body—thus also the pericellular space—was less. Structural details were plainly defined. Pale cells and vacuoles were absent.

Briefly summarised, the author concludes that certain chemical hardening media produce variations in the staining capacity of the ganglion cells which are not apparent in the cells of the fresh cord—Müller's fluid acting with special potency in this way. Whether the effect in reality constitutes a micro-chemical test, of far-reaching import in connection with the anatomical and physiological relationships of the affected cells, is yet an open question.

As neither pericellular spaces nor vacuoles were found in fresh tissue, it may be assumed that their presence in certain of the hardened specimens was due to the chemicals used.

The changes caused by alcohol are less disturbing and not so liable to lead to erroneous conclusions as those produced by chromic acid or its salts. They are in no way more pronounced when absolute alcohol is used from the commencement than when a weak mixture is first employed.

The examination of the spinal cord of the animal poisoned by phosphorus gave entirely negative results, so far as any specific histological change is concerned.

Heusser on a Case of Lymphosarcoma of the Hypophysis Cerebri. (*Ibid.* Bd. ex. H. 1.)—The patient, a female, aged 64, came under treatment for cancer of the rectum. Four weeks prior to admission left ptosis commenced; two weeks later it had become complete. Concurrently with this palsy, violent pains in left side of face, radiating to the vertex, set in, and visual power in the left eye became considerably impaired. The headache, dizziness, and nausea, which had troubled patient for some time, were attributed to the great exhaustion induced by the rectal disease. A palliative operation in connection with the latter ended in fatal collapse.

A reddish tumour of moderately firm consistence, about the size of a prune, was found occupying the sella turcica, the left cavernous sinus and optic foramen, reaching also into the left ectemoidal cells. The tumour was of irregular shape: its base was traversed diagonally by the left optic nerve. Near to the left of the nerve was the ophthalmic vein with pretty wide lumen. The chief mass of the tumour was external to this vein. The base of the tumour surrounded the internal carotid artery, the lumen of the latter being scarcely diminished. The left third, fourth, and sixth nerves were also embedded in the growth, the first mentioned being compressed into a quadrangular shape.

The tumour was mainly formed by uninuclear round cells of nearly uniform size. The nuclei were round, or here and there oval, and very rich in chromatin. Each nucleus almost filled its cell, the protoplasm of the latter being very scanty. Around the vessels, lying amongst these lymphatic elements, were small round or spindle-shaped cells which stained more deeply than the former, and were regarded as granulation tissue cells. The cell masses, which in many places had a follicular arrangement, were surrounded by a delicate and extremely vascular reticulum. In all parts of the tumour numerous red blood-corpuscles filled the small vessels or lay scattered free in the tumour mass. None of the normal structure of the hypophysis was visible.

The sheath of the optic nerve was invaded by the neoplastic cells. Around and within this sheath there also were inflammatory elements, migrated leucocytes, disseminated through the whole nerve, but in greatest number at the periphery.

Besides masses of red blood-corpuscles, very numerous round

cells lay within the internal carotid and the ophthalmic vein. The intima of the former was here and there displaced by subjacent accumulations of those cells: at one part, a thrombus partly occluded the vessel.

The presence of osseous lamellæ in the midst of the neoplasm—entirely surrounded by it—pointed to erosion and exfoliation caused by the proliferous tumour elements.

In addition to the inflamed condition of the optic nerve, Weigert's method showed marked degeneration of its peripheral fibres: at some points the latter had almost disappeared, being replaced by connective tissue. Corpora amylacea were found in the nerve, especially at the periphery.

Lymphatic cells were found in the nerve sheaths of the corresponding third, fourth, and sixth nerves, more particularly where the nerves were compressed by the tumour. A few corpora amylacea could be seen in these nerves, no other degenerative change. E. Bibt.

Eppinger on Miliary Aneurisms of the Cerebral Vessels (*Arch. path. Anat.* Bd. cxi. Hft. 3).—This paper is a reply made by the author to critiques (respectively by Zenker and Lowenfeld) on the appendix to his recently published work on the Histogenesis and Etiology of Aneurisms. In these he was charged with an unauthorised attack on the views of Charcot, Bouchard, and Zenker regarding miliary aneurisms and their relation to spontaneous cerebral hæmorrhage. Professor Eppinger pleads in justification that he has been misconstrued by his reviewers in regard to a point on which he has laid great stress throughout his entire work, viz. his exposition of the nature of an aneurism, and which is one that does not allow him to accept that described by Charcot, etc., as such according to his definition. Extended research compelled the author to emphasize the need of a greater restriction as to the meaning of aneurism. His diagnosis of the true aneurism comprised essentially, the interruption of the elastic coat at the entrance to the swelling, its absence from the wall of the same, and the continuous transit of the remaining vessel-wall, entire or isolated, leading to or from the aneurism. Hence he rejects simple ectasie, in which, changed or unchanged, the laminæ are continued over the swelling, dissecting and spurious aneurisms. Being convinced that the changes, a part of aneurism as accepted by him, are accomplished in a three-fold manner, he sets forth three forms of such conditions: (*a*) con-

genital, (*b*) parasitical (including mykotische-embolische, equiverminosum, and arrosious aneurysma occurring in pulmonary tuberculosis), and (*c*) simple, traumatic. In these, he has been able to comprehend all known aneurisms possessing the required characters. He insists especially on his definition of aneurism as representing a fixed and, histologically, well-marked disease of the artery; and whilst perfectly recognising the miliary aneurisms of Charcot and Bouchard, he has excluded them from his category as not conforming to such definition, discarding both them, as simple ectasies and perivascular changes. Atheroma as a basis of moment for the production of aneurism he also discountenances. Finally is pleaded that, not until the standpoint assumed by the author, regarding the fixed nature of aneurism, can be rendered untenable by indubitable examinations and pathologico-anatomical inferences from the same, should such harsh criticisms as have been suffered by him be justifiable. There is much interesting matter in Professor Eppinger's paper, but unless viewed in conjunction with his original work, not to hand, it would be of little service to produce it. F. St. John Bullen.

Haig on Headaches, Fits, and Mental Depression due to the Abnormal Uric-Acid Elimination.—Dr. Haig has pointed out (*St. Bartholomew's Hospital Reports*, vol. xxiii. p. 210) the close clinical relationship of epilepsy to a form of headache, which he identifies with migraine, and believes to be due to the uric acid in the blood; and has shown (*Neurologisches Centralblatt*, March 1st) that the fits in some cases of epilepsy are accompanied by an excessive excretion of uric acid exactly resembling that which occurs in the uric-acid headache. See also papers on the excretion of uric acid under various drugs (*Medico-Chirurgical Transactions*, vol. lxxi.) and Thesis for the degree of M.D., Oxford (abstract in *British Medical Journal*, vol. ii. pp. 10, 88). Dr. Haig believes that such cases of epilepsy may be successfully treated by diet or drugs which promote the elimination and prevent the retention of uric acid in the body.

In several of these papers he has mentioned some interesting symptoms which serve, he thinks, to link headache, epilepsy and mental depression to each other, and also to gout.

He has also pointed out in the above Thesis, and more recently (*Practitioner*, November 1888), that mental depression is often due to excess of uric acid in the blood, and that it may be controlled and successfully treated by acting on the uric factor

That there is an intimate connection between uric acid excretion and this mental condition is shown by, *inter alia*, the fact that when a plus excretion of uric acid is going on under the influence of an alkali, there is depression, bad temper, and lessened mental activity. On the other hand, when there is a minus excretion of uric acid under the influence of an acid, there is a bright and cheerful feeling with increased mental activity; excessive excretion meaning, the author believes, excess in the blood, and *vice versâ*.

Many people suffer from some amount of mental depression during the "alkaline tide" of the morning, when the excretion of uric acid is normally at its highest point; and mental depression may be caused in many people by giving alkali to produce an artificial alkaline tide.

The fits in some cases of epilepsy are preceded by a minus excretion (or retention) of uric acid, and are accompanied by a plus excretion of uric acid; and it has been observed that some patients are able to foretell the advent of their fits by the bright and joyous feelings which correspond, as there is no doubt, to the retention preceding them. A precisely similar fluctuation in mental conditions has been noticed by Haig and others in the case of the uric-acid headache, no doubt having a precisely similar causation.

An attack of melancholia may terminate suddenly in an attack of gout, and if melancholia is due, as Haig believes, to uric acid in the blood, a dose of acid in driving it out of the blood into the joints will cure the mental and produce the bodily disease in a way that it is very easy to imitate; for any one who is suffering from a uric-acid headache can cause pricking pains in his joints, more or less severe, by driving it out of his blood with an acid; and the same dose of acid, if given to a gouty patient, will at once make the joint pains worse.

Further, the author considers it to be a very important distinguishing mark of the mental depression due to uric acid, that in those who have joint pains it does not occur along with the pains, but alternates with them; thus a gouty patient says: "I had gout two or three years ago; now the pains have gone, and I have depression and heaviness worst in the morning." He is in poor circumstances, or otherwise run down; if you can improve his diet and nutrition, and so raise his acidity, you will bring back his gout and remove his mental depression.

Many patients who suffer from uric-acid headache suffer also from some mental depression, which can be cured in the same way

as the headache, by a dose of uric acid; their place being taken by bright and happy feelings of well-being.

Dr. Broadbent has pointed out that a high-tension pulse is a very constant symptom in cases of melancholia; and Haig has remarked that the pulse tension during the uric-acid headache is generally very high, and that, *cæteris paribus*, pulse tension varies with the amount of uric acid present in the circulation, so that it is possible, by washing an excess of uric acid into the blood with an alkali, to produce headache, or mental depression and increased tension of pulse, the excretion of uric acid in the urine rising *pari passu* with its increase in the blood; or if these conditions are present, the headache or depression may be driven away, the pulse tension reduced almost to nothing, and the excretion of uric acid in the urine diminished by giving an acid. This fact seems to prove that the mental depression, and the high-pulse tension which accompanies it, are both due to the excess of uric acid in the circulation.

The treatment of such conditions is that of gout and of the uric-acid headache: a diet poor in nitrogen; or drugs, such as salicylate of soda, which eliminate uric acid. This drug, when given for gout or epilepsy, often has a marked effect in improving the spirits; and it differs in this important respect from an alkali; that while causing plus excretion of uric acid, it yet causes no headache or mental depression.

T. W. Shore on the Morphology of the Vagus Nerve.—(*Journal of Anatomy and Physiology*, vol. ii., part 3, April 1888.) The Author prefaces his remarks on the morphology of the vagus, by summarizing the leading features of its anatomy and development, in petromyzon, elasmobranch fishes, and rana, together with its development in the chick. He remarks that if Gaskell's researches, on the visceral nerves of the dog, hold good for lower types, a perfect spinal segmental nerve must contain somatic motor, somatic sensory, and splanchnic (motor and sensory) fibres, and that inasmuch as the vagus in amniota (with the exception of the small dorsal branch), as well as in fishes (excepting the lateralis and the afore-mentioned small dorsal branch), is wholly visceral in distribution, the current hypothesis that it (the vagus) is a compound of a variable number of *segmental* nerves is untenable. He regards the visceral portion as representative of the coalesced visceral rami, corresponding to the anterior end of the alimentary tract, and including the branchial nerves of fishes. He supports

Hubrecht in identifying the ganglia of the vagus, and other cranial *nerves*, with the brain lobes, of the nemertea, and further thinks that they probably also represent the cerebral ganglia of annelida and arthropoda. The ramus lateralis he regards with Hubrecht to be identical with the lateral nerve strands of nemertea, and, in addition, regards it as being homologous with the main nervous system of annelida and arthropoda, and possibly also with the cœlenterate nerve ring. The study of the vagus may, in his opinion, throw light on the question of the chordate ancestor, but will not help to any great extent in solving the problem of the segmentation of the vertebrate head. In conclusion, he suggests that the following points should be elucidated on the plan initiated by Gaskell on the dog, *i.e.* by studying the histological composition of the various parts of the vagus.

1. Does the "ramus visceralis vagi" resemble in structure the visceral portions of spinal nerves?

2. Are the fibres of the branchial nerves of the same nature as those composing the "ramus visceralis"?

3. Do the fibres of the "ramus lateralis" resemble histologically those of the rest of the nervous system?

4. Do the spinal metameric nerves of the segments corresponding to vagus visceral distribution, themselves contain visceral fibres?

With these questions he hopes to deal in a subsequent paper, to which this one must be regarded as a preface.

W. L. Symes.

Ball on Traumatic Aphasia relieved by the Removal of a Blood-clot from the Cerebrum (*Dublin Journal of Medical Science*, September, 1888).—The author records a case of peculiar interest successfully operated on by him for the cure of aphasia. The patient had been struck a blow in the head with a penknife ten days before admission to the hospital. He had noticed, since the blow, increasing difficulty in using the right words. On examination, a small scab was found adherent to the scalp, over the squamous portion of the left temporal bone. This, when detached, showed a cicatrix extending deeply through the temporal muscle. There was well-marked motor aphasia, word-blindness, and word-deafness. No paralysis could be detected. Five days after admission, as the symptoms had increased, an operation was determined on. A flap was turned down including the cicatrix, and a wound of the squamous portion of the temporal bone, such as could be caused by the small blade of a penknife, was found. A circle of bone was removed, containing in its centre the wound in the bone. The knife was found to have penetrated the dura mater and brain. The dural wound was enlarged and a Sims' forceps was passed into the brain wound, and was gradually separated. A dark-coloured blood appeared and was extruded by the internal brain pressure; more clot was removed by Sims' forceps and weak perchloride irrigation, a drainage tube was introduced, and the external

wound was sutured. On the evening of the same day the patient carried on a long conversation with very few mistakes in his selection of words. The following morning aphasia was again increased, but disappeared on cleaning the blocked drainage tube. The recovery was, after this, uninterrupted and complete.

J. W. White, M.D.

J. Hoffmann on Tetany (*Deutsch. Arch.*, 1888, xliii. 53).—This paper is based on eleven cases of tetany, reported in full, and accompanied by numerous references to cases of other observers and their opinions concerning the disease. In all but one of the author's patients the affection began between the ages of sixteen and twenty-five years. He is disposed to believe that the nature of the occupation is not without influence on its etiology, and that shoemakers, seamstresses, clerks, &c., are, perhaps, especially liable to it. After reviewing at some length the literature relating to the bearing of the thyroid gland on tetany, he concludes that, in all probability, the removal of this organ is one of the etiological factors. He does not agree with Strümpell that they are endemic influences in its production. The spasm was rather widely spread in the cases which he reports, and was always bilateral. The attacks lasted from several minutes to some hours, and in one instance ten days. The premonitory fibrillar muscular trembling was absent in most cases; the tendon reflexes were usually normal, but in one case diminished, and in another nearly absent. The attacks of spasm were always preceded or accompanied by paræsthesias. Trousseau's symptom (the production of an attack by compression of the main artery of a limb) was always present during the height of the disease, while the compression of a nerve had this effect in but one case. The disappearance of this symptom is in no sense a sign that the disease has terminated, but only that it is in abeyance. The galvanic and faradic electrical excitability of the nerves was much increased in all cases in which it was carefully examined; and even the facial and the hypoglossal nerves may share this excitability. An increase of the mechanical excitability of the motor nerves was absent in but one instance. A series of experiments convinced the author that the mechanical and electrical excitability of the sensory nerves is increased. The disease lasted in the author's cases from half a year to twenty-one years. Most commonly the attacks occur in groups, lasting some weeks, and with variable periods between them, in which there exist only certain symptoms of latency; or the intervals may continue for years, with absolutely no sign of the affection.

According to most authorities, the prognosis is favourable, though the disease may last for years. Death occurs only exceptionally, from exhaustion or spasm of the diaphragm. Certain of the author's cases exhibited evidences of paralysis, for which the tetany was apparently responsible. Frequent shedding of the finger-nails and a brown pigmentation of the skin of the hands and face was observed in one instance, and the author considers the probable cause of the latter to be nervous influence combined with repeated small extravasations of blood into the skin, induced by the muscular spasm. Hoffmann reports the results of autopsies which have been made, and quotes extensively the opinions of various writers concerning the pathological anatomy of tetany; all of which indicates that, though fine molecular nutritive changes of the nervous system are almost universally considered to be the cause of the disease, making it, therefore, a neurosis, the seat of the changes is still a much disputed point. He obtained the best therapeutical results from bromide of potash, morphia, and the galvanic current. In an appendix the author reports four additional cases of the affection, in two of which shedding of the nails took place, accompanied, in one instance, by falling out of the hair. He shows still further the positive influence of extirpation of the thyroid gland on the production of tetany, and calls attention

to the fact that attacks seldom occur in summer, cold seeming to be a strongly exciting factor.

Oppenheimer on Railway Spina (*Dublin Journal of Medical Science*, October 1888).—Certain symptoms are common to all, or at least to the majority of cases of nervous disease consequent on spinal concussion without external injury. These symptoms are chiefly psychic, and belong to the affection, mental depression and irritability occupying the foreground. The mental depression is accompanied by anxiety of mind, the patient's thoughts being always occupied, even in his dreams, with the accident, but is distinguished from pure melancholia both by the abnormal irritability always present, even in persons previously remarkable for their phlegmatic indifference to internal excitations, and by its hypochondriacal character. Severe intellectual disturbance is rare, but the author has seen a considerable degree of dementia with weakness of memory, also cases of hallucination, and even of traumatic insanity, requiring seclusion. Giddiness and cramps, varying between "petit mal" and true epilepsy, are not infrequent; but care must be exercised in diagnosticating such from purely hysterical attacks.

In the domain of the special senses, a mixture of hyperæsthesia and anæsthesia, or rather their juxtaposition, is the most frequent characteristic—*e.g.* hyperæsthetic zones are found in the anæsthetic cutaneous areas; acuteness of vision is lowered, while the eye is extremely sensitive to light; the auditive faculty is lessened on the whole, while certain sounds react abnormally. Sensory anæsthesia is a very important subject, and observations have been made in Westphal's clinic with a view to distinguish genuine from simulated symptoms. The anæsthesia, *e.g.* of an extremity, does not usually follow the course of the nerve implicated, but is spread over neighbouring nerves, and is often overlooked. Typical girdle-feeling, and a corresponding anæsthetic zone, are rare. Pains are frequent, and usually of the dull kind, especially headaches. Reflex excitability is more often lessened than increased. The tendons always react. Motility is almost always affected; the patients move about slowly, and without energy; the spine is maintained as fixed as possible, sometimes reflex muscular symptoms hinder locomotion. The various ways of walking are remarkable, and may excite suspicion of simulation. There is no doubt that the usual signs here do not, as a rule, correspond with those dependent on "material diseases" of the central nervous system. When one foot drags, it is not carried as in hemiplegia. Tremor is frequent, but it resembles that of hysteria far more than that of sclerosis. Swaying on closure of the eyes is very frequent. As to the motor cerebral nerves, the speech suffers most, though there is no trace of aphasia. Speech is irregular and interrupted.

H. C. Tweedy, M.D.

END OF VOL. XI.

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